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TO

THOMAS BARLOW, M.D. (LOND.), F.R.C.P.,

PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL, AND TO THE CHILDREN'S HOSPITAL, GREAT ORMOND STREET,

THIS WORK IS DEDICATED

In Admiration of

HIS POWERS OF OBSERVATION, HIS CLINICAL ACUMEN, AND HIS SOUND JUDGMENT;

AND

In Gratitude for

HIS TRUE-HEARTED FRIENDSHIP, WHICH THE AUTHOR HAS ENJOYED FOR MANY YEARS.

PREFACE.

THE chief aim of the present manual is to assist the student and junior practitioner in the Examination of Medical Cases. For this reason, symptoms and signs are treated of rather than diseases, and much thought and labour have been devoted to the arrangement of symptoms in the order in which they are usually considered in practice. No arrangement can be perfect in this respect, for no two cases are examined in precisely the same way. Nevertheless, it is hoped that the method adopted will impress upon the student the necessity of eliciting *facts*, and of recording them in a correct and orderly manner, and that it will also be successful in giving him a firm grasp of the subject of Clinical Medicine.

A large number of the illustrations are from photographs or drawings of cases under my own care; for others I am indebted to the kindness of some of my colleagues, while the remaining illustrations have been collected from various sources, which in all cases are duly acknowledged.

In the preparation of this volume I have received help from Dr. Williamson in the sections relating to the blood, sputum and puncture fluids, and from Dr. Kelynack in those on the pulse and temperature. I am also greatly indebted to my friend and colleague, Dr. Harris, for his kindness in writing the section on Laryngoscopy, and to Dr. Wild for that on skin eruptions; while to my friends Dr. Barlow, Dr. Dixon Mann, and Dr. Steell, I owe thanks for a number of valuable suggestions and corrections.

My warmest thanks, too, are due to Dr. George Lang, of Bromley, for the Index and Table of Contents which he has so ably prepared, and for the great assistance he has given me in proof-reading. Dr. Lang has read and criticised every page of the proofs, and it is to his ability and care that many omissions have been supplied and many mistakes and inaccuracies corrected or avoided. Finally, I must express my acknowledgments to the publishers for their kind assistance in many ways, and especially for the liberality with which they have met my wishes in regard to the illustrations.

JUDSON S. BURY.

10 St. John Street, Manchester,
April, 1894

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CHAPTER I.

INTRODUCTORY.

It is difficult or perhaps impossible to give an accurate definition of either health or disease, for there is no sharp boundary-line between them. Disease implies a derangement of the healthy functions or structure of the body, and it may exist for some time before leading to recognisable manifestations of its presence. The actual onset of diseased change in function or structure is indeed for the most part unknown to us during life, for its manifestations only appear at a variable period of its development; these are called **symptoms**, and must be regarded as integral parts of the disease. Recognisable departures from health, which are perceived only by the patient, are called **subjective symptoms**, and these frequently he endeavours to express by words or gestures. Recognisable departures from health, which can be detected by the senses of the observer, are called **objective symptoms**. Some writers call the former simply "symptoms," and the latter "physical signs." Others give the term "symptoms" a wider application, and "physical signs" a narrower one. Thus under "symptoms" they include not only the subjective manifestations of disease, but certain objective symptoms, such as pyrexia, emaciation and vomiting, while they restrict the use of the term "physical signs" mainly to the results obtained by an examination of the chest and abdomen and nervous system. Certainly such a distinction is of considerable importance in the study of disease. For example, two patients present identical physical signs of phthisis—impaired resonance, harsh breath sounds, and a few moist râles at the apex—but while one of them is very ill, the other is comparatively well. The former patient suffers from pyrexia, emaciation and much weakness, whereas the latter is quite free from any general manifestations of disease. Moreover, the symptoms just referred to may all subside, while the physical signs remain much as before. Another example is mitral disease—how varied are the symptoms in different cases in which the physical signs, so far as palpation, percussion and auscultation go, are almost identical. Similar observations might be made in respect to many other diseases. Symptoms, indeed, have a cyclical history of their own, and their

relation to the few coarse physical signs which we are able to make out is often much too subtle for our ken. At the outset, then, let the student fully realise that symptoms really follow a course of their own, apparently quite apart from the signs which are revealed to him by the various methods of physical examination.

Further, it is to be noted that the severity and significance of subjective symptoms vary greatly in different individuals. A man of strong constitution may become the subject of grave disease without knowing that anything is amiss, while a delicate person with susceptible nervous system may feel the slightest departure from health. In the latter case, subjective symptoms are of great value and require careful study, although, at the same time, their importance may easily be exaggerated. For the investigation of objective symptoms, the unaided senses in many cases are sufficient, while in other cases the employment of certain instruments, such as the stethoscope or ophthalmoscope, or of chemical tests, is necessary to complete the examination.

Of the various methods of examination, which will be explained in due course, a thorough inspection of the patient is of very great importance. It is necessary to emphasise this fact, because this mode of inquiry is so often neglected by the student. It is remarkable how much valuable information may be obtained by the trained eye from a careful general inspection of the surface of the body; indeed, many diseases can be diagnosed by this method alone; and although it would be foolish to neglect making a thorough examination of the patient by every other means at our disposal, we would insist on the great importance of subjecting to careful analysis and consideration not only the more obvious signs of disease, but also those more subtle signs which may be revealed by the expression of the features, the posture of a limb, or the gait in walking. These signs convey impressions to our minds which it behoves us to sift and weigh, for they often have an important bearing on the diagnosis and prognosis of the case. Indeed, it is not a rare experience to hear of the death of some one in whom the physical signs were never marked or of serious moment, and afterwards one says, "Ah! I remember how ill he looked." If this impression, unconsciously received at the time of examination, had been recognised and its meaning sifted, it would have led to more repeated examinations; possibly we should have been able to place the patient under more favourable conditions, or at any rate could have warned the friends as to the probably dangerous nature of the malady.

The method of conducting the examination of a patient will obviously vary to some extent with the nature and severity of the illness, but in all cases it is necessary to take into consideration :—

(1.) The symptoms for which the patient seeks or is brought to us for advice.

(2.) The account of his illness as given by the patient or his friends, together with facts relating to his previous health, habits, place of residence, occupation and family history.

(3.) The symptoms observed in making a physical examination of the case.

Some physicians prefer to examine a patient before giving ear to the history of his complaint, but the author believes that it is much better to take the latter first and to listen attentively, and as far as possible without interruption, to the patient's own statement of his case. When the patient is unconscious, very young, or otherwise incapable of giving an account of himself, then we have to depend on the statements of his friends.

While listening to the account of the illness, we often unconsciously receive impressions from the attitude, general aspect and psychical condition of the patient, which subsequently may prove to be of great help to us in forming an opinion with regard to the diagnosis, prognosis, and treatment of the case.

In reporting cases, special prominence should always be given to the symptom or symptoms for which the patient seeks advice. The complaints of the patient form the text of the case, and should be referred to again and again during the examination. Sometimes they appear to have no connection with the results of a physical examination, but often there is a direct relation between the two, and it is certainly of the greatest importance never to lose sight of the patient's own words with regard to his trouble. These should be given in inverted commas, and the order in which the symptoms are related should be preserved as far as possible, and on no account must the words of the patient be translated into scientific terms. His version as to the cause of his illness, and any evidence that he has to give in support of his view, should be recorded. Gaps in the history may be filled up by inquiries as to different symptoms. The first sign of ill-health and the date at which it occurred should be especially noted, also the date at which the patient left off work and the date when he took to bed. But the information obtained by more or less leading questions should be distinguished from that which is volunteered by the patient. With regard to the former, there is an occasional danger of fallacy owing to the patient wishing to make his case out worse than it really is.

Inquiries should also be made as to previous attacks of the same kind of illness, and as to the previous health generally. In recording past illnesses, corroborative evidence of their existence should always be asked

for. For example, it is not enough to say that a patient had syphilis or rheumatism. But statements should be made, in the case of supposed syphilis, as to the presence of a sore followed by bad throat and "breaking out" on the skin so many weeks afterwards, and by pain and swelling of the shins so many months afterwards; while in the case of supposed rheumatism, inquiries should be made as to the length of time in bed, the presence and duration of sweating, and the number of joints affected with pain and swelling.

Questions should then be asked as to the existence of any similar or allied diseases amongst the patient's relatives. But it is necessary to observe that with hospital patients there is a great risk of fallacy in these family details.

Having obtained a full account of the previous history of the patient, the next step is to make an examination of his present condition, "to take the present state" as it is sometimes called. At the outset we should ask ourselves is there any striking dominant symptom? If there is, we should begin with the system to which that symptom belongs; thus if the patient is paralysed, we begin with the nervous system; if he suffers from shortness of breath and dropsy, we begin with the circulatory system, and so on. If there is no striking dominant symptom, it is well to return to the patient's own complaint, and investigate that and the system with which it is connected. But with whatever system we begin, it is necessary to make a thorough examination of the whole body, and to draw up a report as to the condition of its various tissues and organs.

As a guide to the principal subjects of inquiry, the following form, which is now in use at the Manchester Royal Infirmary, is appended:—

DIRECTIONS FOR REPORTING MEDICAL CASES.

PRINCIPAL SUBJECTS OF INQUIRY.

PREVIOUS HISTORY.

1. *History of Present Illness.*

Mode of Onset.—The first sign of ill-health—the date of month when patient left off work, and date when he took to bed.

Course of Symptoms.—Give order of occurrence, with date of commencement and cessation. The condition of the patient at the time of admission, and the course of the case to the time at which the notes are taken should be stated.

Supposed Cause of Illness.—Give patient's version, *treatment before admission*, and its effects (briefly).

2. *Previous Health.*—Nature and character of previous illnesses, their dates and duration—(ailments of infancy in special cases)—previous admissions into hospital—indications of gout, rheumatism, venereal diseases (gonorrhœa, soft chancre, hard chancre, sore throat, pains in bones, &c., symptoms of infantile syphilis, if necessary)—cough—hæmoptysis.

Sexual disorders—catamenia—leucorrhœa.

Inquire specially for previous hemorrhages or discharges, if there be anæmia.

Previous general nutrition and weight.

3. *Social History.*—Particulars concerning residence, coldness, dampness, salubrity—prevalence of special diseases—changes of residence—residence abroad.

Occupation—peculiarities of occupation, as exposure to heat, cold, noxious gases, dust, &c.—changes in occupation.

Food and clothing—excess or defect, &c.—stimulants, character and amount—tobacco—drugs.

General or special habits and mode of life.

If married, date of marriage—issue—still-born children—miscarriages and times at which they have occurred.

4. *Family History.*

Father, Mother, Sisters, Brothers, } Ages, health, or cause of death.

Diseases and causes of death in other relatives, especially consumption, insanity, gout: specify whether relative affected was on side of father or mother.

PRESENT STATE.—GIVE DATE.

(*a.*) *External Surface.*—Posture—temperature—general appearance—form of head—colour and expression of face, worn, languid, sallow, excited, stupid, livid in any part, flushed, anæmic—wrinkles—nostrils—lips—arcus senilis—conjunctivæ—pupils—eyelids—eyeballs—ears.

Relation of appearance to age. Nutrition—well nourished, stout, spare or emaciated, weight.

Peculiarities of external configuration, tumours, swellings, deformities.

Skin—œdema, face, body, ankles, &c. Perspiration—face, head, body—odour, if any—cutis anserina—roughness—softness—cicatrices—rashes (maculæ, stains, erythemata, wheals, papules, vesicles, pustules, scaliness, tubercles), ulcers—discolourations and superficial vessels.

Hair on head, body. Nails curving—clubbing of fingers—onychia—structural alterations.

Glands—superficial, back of neck, along sterno-mastoid, in axilla, groin—parotid.

Joints—form—redness—tenderness—pain—stiffness—deposits around—deformities.

(*b.*) *Nervous System.*

1. *Motor Disorders.—Spasm.*—Tonic, clonic, distribution of, whether general, unilateral, or local, distortions resulting from spasm. *Tremor* and its relation to voluntary movement.

Paralysis.—Spastic or atrophic, and distribution of, whether in the form of hemiplegia, paraplegia, hemiparaplegia, or local, distortions of paralysed limbs.

Station.—With and without closed eyes.

Gait.—Whether ataxic, spastic, paralytic, shuffling, staggering.

Strength.—As tested by ability to move in bed, by power of grasp, and of resistance to passive movements at the various articulations.

Disorders of special movements, caused by paralysis of the ocular motor muscles, the facial, lingual, and masticatory muscles.

2. *Sensory Disorders.*—Acuteness of cutaneous sensibility, as tested by pricking, pinching, touch, double points, hot and cold bodies, faradism and different weights; change of position on the eyes being closed; touching prescribed spot on face: pain—sense of constriction, &c., paræsthesia, such as numbness, tingling, &c.

Spine.—Tenderness, hyperæsthesia to pinch or to faradism.

Distribution of anæsthesia, whether hemianæsthesia, paranæsthesia, local anæsthesia.

3. *Reflexes.*—Superficial, deep.

4. *Vaso-motor* and *trophic* disorders—redness or pallor of surface, temperature of surface, cutaneous eruptions, glossy skin, deformities and cracking of nails, abnormal dryness or premature greyness or falling off of the hair.

5. *Electrical Reactions.*—Faradic, galvanic.

6. *Psychical Disorders.*—Emotional condition, irritability, insomnia, vertigo, incoherence, depression or exaltation of spirits, hallucinations or illusions of the senses, delusions.

(*c.*) *Circulatory System.*—Palpitation—dyspnœa—syncope—pain, its character.

Heart.—Cardiac region, general appearance, normal or bulging; impulse, its area, strength and character (normal, heaving, weak), apex beat—note the intercostal space, distance from left vertical nipple-line, or *especially in women*, distance from mid-sternum—if thrill or friction fremitus—note its site and rhythm.

Percussion.—Map out superficial cardiac dulness—give upper limit, right limit, left limit of deep dulness. Note any prominence or dulness of mediastinal region.

Auscultation.—Note character of *sounds* at apex, over ensiform cartilage, at aortic and pulmonary cartilages, accentuation, loudness, reduplication, or other special characters. If *murmurs* or *friction*, give rhythm, situation of maximum intensity, and the direction in which they are conducted.

Arteries.—Undue pulsation of carotids and brachials—any abnormal pulsation above manubrium or above clavicles or elsewhere. If a tumour be present, note if pulsation is expansile, any thrill—tortuosity of arteries at elbows and over temples.

Radial Pulse.—Compressibility, regularity, frequency, size. Are the two radial pulses alike? Sphygmographic tracing.

Veins.—Undue visibility on chest wall—pulsation in neck. Do jugulars fill from below? Varicose veins and venules in different parts of body.

Capillary Pulsation.

Examination of Blood.—Numeration of corpuscles by hæmacytometer, estimation of hæmoglobin.

(*d.*) *Digestive System.*—Lips—teeth—gums—tongue—fauces and pharynx—deglutition—thirst—appetite—abdominal pain or discomfort, its relation to the taking of food, to defæcation, to movement, micturition, &c., flatulence or acidity—nausea, vomiting, SEE *vomited material and describe*—frequency of defæcation—character of stools, SEE *stools, if possible, and describe*—hæmorrhage—piles—pruritus ani, &c.

Physical Examination of Abdomen—shape, measurement at epigastrium and at umbilicus—condition of umbilicus—aortic pulsation—superficial veins—palpation, thrill, amount of resistance, feel for edge

of liver, for edge of spleen, for mesenteric glands, for kidneys, for enlarged pelvic organs or tumours, and define—*percussion*, define upper limit of liver dulness in right nipple-line, in mid-axillary line, in line of angle of scapula; define splenic dulness, any dulness in flanks, give limits and note change with change of position. If *tumour*, note percussion limits, also its position, alteration of position with respiration, its mobility, the character of its surface, its consistence, &c. Presence of hernia.

(*e.*) *Respiratory System.*—Cough, character of; expectoration, character of, presence of blood, microscopic examination—elastic fibres, tubercle bacilli. Pain, tenderness, decubitus, number of respirations, character of, easy, laboured—dyspnœa, inspiratory, expiratory. Give ratio of pulse to respiration. Is there any movement of extraordinary muscles of respiration? Is there any retraction of chest wall during inspiration? Stridor. Voice—examination of larynx.

Physical Examination of Chest.

Inspection.—Shape of chest, depressions or bulgings, want of symmetry, muscular wasting, immobility of inter-spaces; direction of ribs, costal angles. Movements, character and amount, want of symmetry, whether abdominal or thoracic.

Palpation.—Again note difference of movement on the two sides, &c., also vocal fremitus—and relative intensity on the two sides—rhonchal fremitus—friction fremitus.

Percussion.—Relative resonance, deficiency or excess of resonance. Note pitch—high or low; duration—long or short; quality—hard, tubular, &c.; resistance—normal, diminished, increased.

Auscultation.—Changes in character of breath sounds in regard to the duration and intensity of the inspiratory and expiratory portions, especially noting the *quality*, whether of the vesicular or bronchial type, &c. New sounds—dry or rhonchi, moist rhonchi or râles—simple, or with special character—friction sounds, metallic tinkling, &c.

Vocal Resonance.—Increase or diminution of, bronchophony, pectoriloquy, ægophony.

Succussion.—Splashing sounds, &c.

(*f.*) *Special Sense Disorders.—Smell*—hyperosmia, anosmia, hallucinations of smell.

Vision.—Acuteness of, colour vision, hemiopia, polyopia, field of vision, ophthalmoscopic examination.

Hearing.—Acuteness of, as tested by watch and by tuning-fork. Physical examination of meatus and of throat. Air entering middle ear by blowing nose.

Taste.—Acuteness of, as tested by salt, sugar, bitters, &c.

Speech.—Anarthria or dysarthria, stammering, motor aphasia, word deafness, word blindness, verbal amnesia.

(*g.*) *Genito-urinary System.*—Frequency of micturition—difficulty, pain, its character and locality.

Urine.—Quantity in twenty-four hours, colour, reaction, specific gravity, clearness or turbidity, test for albumin, sugar, and bile—amount of chlorides, of albumin and sugar, if present—percentage of urea in twenty-four hours' urine.

Deposits.—Their general appearance. Microscopic examination—ascertain presence or absence of epithelium, pus, blood, casts of the uriniferous tubes—their character, number, and variety—fatty

point of view it is better to discuss its possible causes and to study it in all its bearings before proceeding to make a physical examination of the stomach and other organs of the body.

Some symptoms are mentioned in this section, because it would be inconvenient to separate them from others which strictly belong to it. Thus coma and delirium can be observed by the physician, but for an account of other disorders of consciousness, such as loss of memory or attention, illusions or hallucinations, we have largely to depend on statements made by the patient or his friends.

CHAPTER II.

SYMPTOMS, FOR THE MOST PART SUBJECTIVE IN CHARACTER, FOR WHICH THE PATIENT SEEKS OR IS BROUGHT TO THE PHYSICIAN FOR ADVICE.

It is convenient to group these symptoms according to the system with which they are most closely related, it being clearly understood that such an arrangement does not necessarily imply disease of the particular system.

I. SYMPTOMS INDICATING DISTURBANCE OF THE FUNCTIONS OF THE NERVOUS SYSTEM.

Pain is probably the most frequent symptom for which a patient seeks advice. Its significance varies greatly, partly according to its locality, but chiefly in consequence of the varying susceptibilities of the nervous system in different individuals. As a rule, the information to be derived from a study of pain by itself is trifling as compared with that to be obtained by a consideration of its associations. It must also be borne in mind that the position of pain does not by any means always correspond with that of the disease; frequently, indeed, it is situated at some distance from it. Pain may be generalised all over the body, or limited to a very small area of its surface. General pains occur sometimes at the onset of febrile conditions, as in influenza, typhoid, acute rheumatism. Localised pains also in some cases may appear to depend on a general blood change, while in other cases they are obviously produced by local disease. Whenever possible, it is desirable to ascertain the exact position of pain, its chief characters,

whether aching, throbbing, shooting, &c., and its relation to time and movement.

The term **neuralgia** implies a severe paroxysmal pain in the course of a sensory nerve or its branches. By many authorities the word is restricted to cases where the nerve disturbance is of (so-called) functional origin; by others it is made to embrace cases of organic origin, as well as those where no definite lesion can be discovered.

Neuralgic pain is shooting, throbbing, gnawing, or boring in character, and of very variable duration. Associated with the spontaneous pain there is usually superficial tenderness of the neuralgic area. This may be diffuse, or more commonly is localised to certain points in the course of the nerve or its branches.

These "tender points" usually correspond to places where the nerve emerges from beneath bone or fascia. Sometimes there is tenderness over a vertebral spine, usually at a spot which corresponds to the origin of the affected nerve. In seeking for a cause of any neuralgia, the possibility of irritation at a distance from, as well as along the course of the affected nerve must be debated.[1]

Pain in the Head.—In asking ourselves what may be the cause of any particular headache, it is well to regard the subject both ætiologically and clinically.

The chief causes of headache may be grouped as follows:—(1.) Disturbance of the intra-cranial circulation, as in heart disease; (2.) Changes in the blood, as anæmia, or a toxic condition produced by alcohol, kidney disease, rheumatism, gout, or the inhalation of certain gases, especially in connection with overcrowded rooms; (3.) Pyrexia; (4.) Disease of the thoracic and abdominal viscera, especially of the stomach and sexual organs; (5.) Diseases of the head, the scalp, the cranium, the brain or its nerves; (6.) Disorders of the special sense-organs, especially the ear and eye.

Such a classification must of necessity be artificial to some extent, for often the causes enumerated act in combination. Thus the headache of pyrexia, while doubtless partly due to elevation of temperature and to vascular congestion, is mainly due to a toxæmic blood state. Also the headache of gastric disturbance may be partly the result of anæmia, partly reflex, and partly toxic in origin.

[1] The importance of studying the situation of pain in visceral disease was insisted on by the late Dr. Ross in his valuable paper "On the Segmental Distribution of Sensory Disorders," *Brain*, 1888. Since then the subject has been further investigated, especially by Head and Mackenzie. Dr. Head in particular has demonstrated that certain definite and constant areas of cutaneous tenderness—each area having a maximal region in which there is pain—may be found in association with various visceral diseases, and that these maximal areas coincide with areas that are marked out by attacks of herpes zoster.

Clinically pain in the head may be studied in relation to its locality, character, time and other circumstances.

Locality.—Headache may be diffused and general, or localised to a particular spot. A general headache is sometimes the result of intracranial disease, as meningitis or cerebral tumour, but more commonly it may be traced to some morbid condition of the blood, as that of rheumatism or gout, or to nervous exhaustion.

A frontal headache is common in pyrexia, and the temperature should always be taken when the headache is not obviously related to functional visceral disturbance. Apart from pyrexia, frontal headache is most frequently associated with stomach derangement, while occasionally it accompanies lung disease. Occipital headache may be caused by constipation, hepatic disorders, ovarian diseases, and sometimes by gastric derangement. The deep-seated pain of neurasthenia is often attended by a sensation of tension about the occiput, and by tenderness to touch in that region.

Pain in the temples is met with in anæmia, hysteria, and neuralgia. In anæmia the pain is usually bilateral, and is accompanied by a sense of bursting of the temples. In hysteria pain may shoot from one temple to the top of the head. In migraine pain usually begins on, and may be limited to, one side of the head, but sometimes the headache is general; hence the term "hemicrania" often given to this disease is inaccurate. The condition of the ear requires careful investigation when there is persistent aching or attacks of neuralgic pain in the neighbourhood, while the bones, especially the mastoid, should be palpated for spots of local tenderness.

Vertical headache also is often neuralgic in character. In hysteria the sensation of a nail being driven into the skull, called "clavus," is sometimes experienced. A feeling of weight or pressure on the vertex, often accompanied by a dull diffuse headache, suggests an overtaxed brain, but it is also complained of by hypochondriacal persons.

The Kind of Headache.—Sharp violent pains occur in neuralgia from any cause. Sometimes they are present during the growth of a tumour, or mark the early period of a meningitis. Dull heavy headaches suggest a toxæmic state, as in uræmia, jaundice, chronic alcoholism, lead poisoning. A throbbing headache increased by movement is characteristic of anæmia; it also often occurs in cases of cardiac hypertrophy, especially when this is associated with gastric disturbance. A pulsating constant headache may be due to an intracranial aneurysm.

Relation of Headache to Time, Taking of Food and other Circumstances.—The headache of exhaustion and inanition is most marked on

waking in the morning, and is relieved by food; that of anæmia comes on in the day, and is worse at bedtime. The syphilitic headache, often very severe, intense and persistent, is particularly characterised by nocturnal exacerbations, which deprive the patient of sleep (but be it noted that sleeplessness in syphilitic subjects cannot always be accounted for by pain in the head). This headache is often accompanied by much irritability and mental depression, while in the severest cases the head is too sensitive to lay on the pillow, and the patient sits up in bed holding his head between his hands.

Excitement and movement improve the headache of hysteria; they aggravate that produced by organic brain disease, also the pain of migraine. The pain of organic brain disease is characterised by its constancy, but it is liable to paroxysmal exacerbations, often of great severity. It varies in character and in position. It may be dull, boring, or rending; it may be general or local. When local, the seat of pain sometimes corresponds to that of the tumour, and there may be local tenderness. Tumours of the cerebellum frequently give rise to occipital pain, but occasionally pain is limited to the frontal region. Hence the locality of a brain tumour cannot be diagnosed with any confidence merely from the position of the pain. An aneurysm of the basilar artery usually produces occipital headache.

It will be gathered from the above observations on headache, that while this symptom may be caused by disease of almost every organ of the body, it appears to be most closely related to alimentary disturbances, to anæmia or to some other condition of the blood. Whenever its aetiology is obscure, the temperature should be taken, the optic discs examined, and the condition of the nervous system carefully investigated.

Pain in the Back may be due to caries of the spine, to disorders of the vertebral joints or ligaments, or to muscular rheumatism, the sacro-lumbar muscles being most commonly affected.

Pain in the Back. This may be general or local, and may be caused by disease of the vertebræ, the cord or its membranes, or the spinal muscles. Sometimes it is a referred pain in consequence of visceral disease. Thus an acute pain in the mid-dorsal region is common in dyspepsia, and a similar pain may be due to a loaded colon, pain in the loins often results from kidney or ovarian disease, while pain over the sacrum is often associated with disorders of the uterus or ovaries. Lumbar pains are very common in the lumbar region. It may be most severe in the early stages of many of the specific fevers, particularly in influenza and smallpox. It is also common in rheumatism when the ligaments are lax, and when there is a tendency to lateral curvature. Aching or

side or front of the abdomen. Intense paroxysms of epigastric pain are not uncommon in locomotor ataxy, and are generally associated with disturbed action of the heart and incessant painful vomiting. These attacks of gastralgia, or **gastric crises**, as they are called, along with lancinating pains in the limbs, chest and back, may occur for many years before ataxic symptoms make their appearance.

Colic is a term applied to spasmodic abdominal pain, of which three chief varieties are distinguished, namely, intestinal, renal and biliary, which are produced by irritation of the intestine, ureter and bile-ducts respectively.

Intestinal colic, due usually to some irritant in the bowel or to lead poisoning, is referred as a rule to the neighbourhood of the umbilicus.

Renal colic, usually produced by the passage or disturbance of a calculus in the ureter, is a severe pain which is felt in one loin and flank, and darts down to the groin and testicle, and sometimes to the thigh or to the chest and back. Sometimes there is an associated disturbance of the colon, especially of the cæcum, which becomes paralytically distended, and there may be attacks simulating the beginning of typhlitis.

Biliary colic, commonly caused by the escape of gall-stones into the cystic and common bile ducts, is an agonising pain which is situated in the right hypochondrium, and shoots up to the right shoulder and back.

All forms of colic may be ushered in by shivering or rigors, and be accompanied by nausea and vomiting, clammy sweat, and other symptoms of collapse.

Pain in the Limbs.—This may be the result of syphilis, rheumatism, gout, or other cause which leads to disease of the bones, joints, muscles, or nerves. It is also to be noted that a pain in the tip of the shoulder sometimes occurs in pleurisy and pericarditis; that shooting pains down the arms are sometimes the result of aneurysm, heart disease, angina pectoris, disease of the posterior roots or peripheral nerves; and that lightning-like pains in the lower limbs constitute one of the earliest and most striking features of locomotor ataxy.

Numbness and Tingling of the extremities often attend the onset of acute affections of the nerves, spinal-cord, or brain. They are particularly common initial symptoms in the various forms of multiple peripheral neuritis, and may exist for some time before objective changes in the cutaneous sensibility can be detected. At the onset of peripheral neuritis, numbness and tingling are frequently combined with alternating attacks of burning sensations in the hands and feet, and coldness and deadness of these parts, together with actual paroxysmal pains in the limbs. In the alcoholic variety severe cramps in the muscles of

the calves and other parts of the limbs, morning retching and vomiting, as well as shortness of breath from cardiac weakness, are also frequent symptoms complained of by the patient.

Vertigo.—Vertigo, called by the patient "giddiness," "dizziness," or "swimming in the head," is a disturbance of the sense of equilibrium. The patient feels as if going to stagger or fall, or that surrounding objects are oscillating or moving in a particular direction. The subjective sensation of movement is sometimes accompanied by actual reeling or falling to the ground.

The symptom is a common one, and according to its associations the following varieties may be distinguished :—

Ocular Vertigo.—This is usually due to weakness of one of the ocular muscles, as the external or internal rectus. Often there is some error of refraction; for example, the strain on the internal recti in myopia may be followed by weakness of these muscles, and then near objects become indistinct, and a sense of confusion and vertigo ensues.

Auditory or Aural Vertigo.—Giddiness may be caused by disease of any part of the auditory apparatus, the meatus, tympanum, Eustachian tube, the labyrinth, auditory nerve or its central connections. When associated with tinnitus and deafness, there is probably an affection of the labyrinth, especially of the semicircular canals, and the condition is called Menière's disease or labyrinthine vertigo. The deafness is central, that is, does not depend on impaired conduction through the middle or external ear; the vertigo, which varies in intensity, may be severe enough to hurl the patient to the ground; it is usually paroxysmal, while the tinnitus and deafness are persistent. The attack may be attended by temporary unconsciousness, and in bad cases is followed by nausea, vomiting and symptoms of collapse.

Whenever vertigo is accompanied by other indications of ear disease, a careful otoscopic examination should be made, and a normal condition of the meatus, membrana tympani and Eustachian tube proved before admitting the existence of primary disease of the labyrinth.

Gastric Vertigo.—Giddiness is often complained of by the subjects of gastric or hepatic derangements, and is commoner in ordinary dyspepsia than in serious organic disease of the stomach. It is frequently accompanied by buzzing in the ears, and may be followed by nausea, vomiting, pallor and faintness. There is no deafness, but it is highly probable that a large number of cases of gastric vertigo are really due to disturbance of the semicircular canals. Still in some cases, as, for example, in gout, there often appears to be a close relation between the taking of food and vertigo.

Vertigo depending on Disorders of the Nervous System.—Giddiness occurs in depression or exhaustion of the brain from any cause, such as

excessive smoking, drinking, anæmia, or mental strain. It is usually slight or moderate in degree, while it is frequently accompanied by emotional disturbance, gastric derangement, palpitation and sleeplessness.

Vertigo also occurs in connection with neurasthenia, epilepsy and migraine. When present in epilepsy, either as the aura of a major attack or as one of the chief symptoms of a minor attack, vertigo is almost always attended by loss of consciousness. Sometimes it is a symptom of intra-cranial lesion, being especially related to disease of the cerebellum and its peduncles. In persons past middle life, with signs of arterial degeneration, an attack of vertigo should suggest the possibility of an apoplectic seizure; indeed, temporary vertigo and slight confusion of intellect may be caused by a small cerebral hæmorrhage. In such cases vertigo usually appears with headache, nausea, or vomiting, and often there is a sense of unilateral numbness or weakness.

Disorders of Consciousness.

Exaltation of the Mental Powers occurs as a premonitory symptom of some forms of insanity or delirium. In chronic alcoholism a stage of mental exaltation frequently ushers in the development of other psychical disorders, and is attended by hallucinations of the special senses. The patient imagines himself to be very rich, or fancies that he hears music, while when he closes his eyes he sees bright clouds or other pictures. But it is noticeable that while the patient is becoming more absorbed in contemplating his own thoughts and feelings, his powers of observation and of attention to business are on the wane. The mental excitement, extravagant acts and elation so often seen in the early stage of chronic alcoholism closely resemble the early symptoms of general paralysis.

Perversions of Consciousness are met with in the various forms of insanity, in the delirium of fever, in poisoning by narcotic drugs, and in many diseases of the brain or its membranes. An **illusion** is a false perception of some sensorial impression received from an actual object, but the error can be detected by the mind and corrected. A **hallucination** is a false perception which occurs without the action of any external stimulus; thus voices are declared to be heard or objects seen in the absence of external realities. Hallucinations of hearing are very common in cases of melancholia and delusional insanity.

A **delusion** is a false belief, a perversion of judgment, and always implies a disordered intellect. Illusions, hallucinations and delusions are the chief factors of **delirium**. The mental state in delirium is similar to that in insanity, but the term is commonly restricted to the acute mental derangement which occurs in many fevers, in organic

alcohol, chloroform; uræmia, *diabetes*; syncope, from failure of the *heart's action*; *head injuries*; functional and organic diseases of the *brain*; *hysteria*; *epilepsy*; multiple sclerosis; general paralysis of the *insane*; *meningitis*; cerebral hæmorrhage, embolism, thrombosis, *tumour*, or *abscess*; hyperpyrexia and sunstroke.

When a *person* is deeply unconscious, it is sometimes difficult to arrive at a diagnosis of the cause of his condition.

In such a case, let the student observe the following points:—The *condition* of the heart and other organs; the temperature; the urine; the *odour* of the breath; the pupils; the results of an ophthalmoscopic *examination*; the *presence* or absence of convulsions, of unilateral spasm or paralysis; and the condition of the reflexes. An important indication of the *depth* of coma is *stertor*, or the snoring noise made by the *patient* in *breathing*. This depends on obstruction to the entrance of air into the *chest*, and is usually produced by the falling back of the *tongue* or of a relaxed soft palate.

II. SYMPTOMS INDICATING DISTURBANCE OF THE FUNCTIONS OF THE RESPIRATORY OR CIRCULATORY ORGANS.

Cough.—*Coughing* is a reflex act excited by irritation of the terminal fibres of the superior laryngeal or some other branch of the vagus nerve. After taking a deep inspiration the glottis is closed and then forced open by a sudden expiratory effort.

As a rule, cough is a result of a morbid condition of some portion of the respiratory tract, but if not obviously produced by disease of the respiratory organs, the possibility of the existence of irritation elsewhere must be entertained, and it is especially important to make a careful examination of the throat—the influence of an elongated uvula, post-nasal catarrh, adenoids, or enlarged tonsils being weighed with the results of a physical examination of the chest. The condition of the ear, of the stomach, and other abdominal organs, may also require investigation.

Without attempting to enumerate all the causes and varieties of cough, we would draw attention to the modifications of its tone produced by local changes in the larynx, by pressure on the trachea, and by functional and organic disease of the brain.

Any impairment of the proper vibration of the vocal cords, as that produced by the presence of false membranes, ulceration or paralysis of the cords, tends to make the cough hoarse and croupy, and may diminish or abolish its tone. Here, too, may be mentioned the peculiar empty ineffective cough of diphtheria and of alcoholic paralysis. When there is pressure on the trachea, as from an aortic aneurysm or a mediastinal

tumour, the cough is hard and metallic in quality, and is associated with stridulous inspiration. The cough also occurs in paroxysms, sometimes much resembling those of whooping-cough, but unaccompanied by the characteristic inspiratory noise of the latter disease. Fits of harassing coughing, if not due to advanced phthisis or to whooping-cough, should always make us think of thoracic aneurysm and mediastinal tumour.

As regards morbid brain conditions, we may note the barking cough of hysteria, and the influence of profound apoplectic attacks and of bulbar paralysis in diminishing or abolishing the act of coughing, and thereby tending to cause a dangerous accumulation of secretion in the air passages.

Shortness of Breath.—When a patient complains that he easily gets out of breath, or that he is short of breath when he moves quickly, it is most likely that he is suffering from anæmia or from heart-disease. Shortness of breath is particularly significant of cardiac insufficiency, of weakness of cardiac muscle; thus it is perhaps the commonest symptom complained of by patients suffering from alcoholic dilatation of the heart. The symptom is largely subjective; the patient feels a need for more air, and objective signs of difficult or hurried breathing may be inconspicuous. But, as a rule, a slightly increased frequency of the respiratory movements is observable. The leading respiratory feature, then, in anæmic and cardiac disease is the patient's own consciousness of disturbed breathing.

The opposite condition, in which increased frequency and difficulty in breathing are apparent to the observer, but scarcely noticed by the patient, occurs in chronic disorders of the respiratory organs, as emphysema and chronic bronchitis, especially when the disease has lasted for many years, and the patient has become accustomed to the hindrance to his breathing.

The chief causes of dyspnœa or difficult breathing, and of alterations in the frequency of respiration, are given in the section on the respiratory system. But we may here point out the importance of regarding these symptoms from a wide standpoint, of taking into account not only the state of the lungs and heart, but also that of the blood and nervous system. If, after a careful investigation, no obvious signs of organic disease can be discovered to explain the occurrence of repeated attacks of severe dyspnœa, it is worth while remembering that the latter may be due to pressure on the trachea, or a large bronchus from an aneurysm, or a deep-seated tumour, and sometimes to a stricture of the windpipe. Nor must it be forgotten that laryngeal spasm, with severe paroxysms of dyspnœa—**laryngeal crises**—may constitute very early symptoms of locomotor ataxia. The attacks often closely resemble

those of laryngismus stridulus, while occasionally noisy breathing is accompanied by a spasmodic cough like that of whooping-cough.

Palpitation.—This means that the patient feels the beatings of his heart, and the consciousness is usually attended by a certain amount of distress or actual pain. When the heart is examined, its action may be found to be quite normal, but generally some change in its force, frequency, or regularity accompanies the subjective sensation. The commonest cause of palpitation is dyspepsia. The symptom is also frequently associated with hysteria, neurasthenia, or other functional nervous disorder. Other causes are anæmia, gout, exophthalmic goitre and organic cardiac disease.

In heart disease palpitation is usually much aggravated by exertion and excitement. In Graves' disease, palpitation, combined with throbbing of the larger arteries and breathlessness, may exist for a long time before the other symptoms, prominence of the eyeballs and enlargement of the thyroid gland, become developed.

But, in whatever way palpitation is started, its degree is much influenced by the condition of the stomach; hence an investigation of the functions of this organ is of the first importance.

III. SYMPTOMS INDICATING DISTURBANCE OF THE FUNCTIONS OF THE DIGESTIVE ORGANS.

The Appetite is diminished or lost (*anorexia*) in many diseases, especially when there is pyrexia or disorder of the alimentary canal. But although **loss of appetite** is a common symptom when inquiry is made with regard to it, it is not often a prominent complaint made by the patient, for it is usually overshadowed in his mind by the existence of other symptoms. Thus a patient attacked with a febrile disorder complains that he feels ill, cold, or weak, or that he has been shivering, or of some symptom peculiar to the particular disease from which he is suffering, such as sore throat in scarlet fever, joint-pain in rheumatism, &c. Perhaps certain forms of dyspepsia and early phthisis are the commonest conditions in which a patient consults a doctor for loss of appetite. In chronic disease, mental depression always requires consideration as a factor leading to anorexia.

Excessive Appetite (*bulimia*) occurs in certain nervous diseases, in diabetes and in some varieties of indigestion. Children suffering from worms or gastro-enteric catarrh are often stated by their mothers to "eat all before them." An **unnatural appetite** (*pica*) for the most peculiar articles is sometimes observed in hysteria, pregnancy, and insanity.

Thirst.—When great thirst and an intense feeling of weakness are

nerves to the medulla. The stimulus may be started at any of these places. First, the centre may be directly affected by a growth, hæmorrhage, or local disease. Poisoned blood may act on the centre, as in acute fevers, uræmia, or from poisons or medicines absorbed into the blood; for example, the vomiting produced by a hypodermic injection of apomorphia. Secondly, vomiting may be started by irritation of the afferent nerve endings to the fauces, pharynx, œsophagus, stomach, uterus, or other abdominal organ. Thirdly, by brain irritation.

Vomiting is an initial symptom of many acute diseases, and this is particularly the case in early life. It is especially noticeable at the onset of scarlet fever, pneumonia and tubercular meningitis, of which diseases it may be a symptom of great diagnostic value. Thus, a young child is taken suddenly ill, the temperature is raised, the fauces are congested and swollen, and there is a slight eruption of uncertain character over the upper part of the chest. Now the occurrence of vomiting on the first day of such an illness is highly suggestive of scarlet fever, but in the absence of vomiting the case may turn out to be one of ordinary sore throat, or of a general catarrh with subsequent localisation in the bronchial or gastro-intestinal mucous membrane. Vomiting is one of the earliest symptoms of acute peritonitis, and also occasionally of pleurisy, pericarditis and phthisis. In elderly persons it often marks the onset of cerebral hæmorrhage; and at all ages it frequently occurs as an early symptom of brain disease, and even when the lesion is one of limited extent.

Vomiting is a striking feature of the paroxysms of whooping-cough, occurring sometimes with such severity and frequency as to place the patient in great danger. During the convalescence of typhoid fever both vomiting and diarrhœa are easily excited by even slight additions to the diet. Vomiting is also not uncommon during the course of phthisis, heart disease, Addison's and Bright's diseases. It is often a prominent symptom in both functional and organic disorders of the stomach and bowels. It occurs frequently in the catarrhs of the alimentary tract that are so common during the period of infancy and early childhood, and may be associated with either obstinate constipation or severe diarrhœa.

Sometimes there is a difficulty in deciding whether a patient is suffering from atonic dyspepsia or gastric ulcer: the occurrence of vomiting in such a case would of itself be in favour of ulcer. But when the ulcer is situated on the anterior surface of the stomach, vomiting and other symptoms may be absent from first to last, or till the onset of perforative peritonitis. In cancer of the stomach, vomiting is most common when the orifices are obstructed, or when there is an ulcerated surface. When new growth is limited to the walls of the stomach, the

stress, too, may be laid on the need of examining the fæces, in order to avoid the risk of overlooking malignant disease, and of remembering that a thin discharge associated with "weeping" often occurs near a partial obstruction.

Constipation is a prominent symptom in many disorders of the heart, liver, stomach and nervous system. It occurs in anæmia, in diabetes and at the commencement of many febrile diseases; it constitutes an early sign of peritonitis and is an important symptom of intestinal obstruction.

In the acute varieties of obstruction, constipation is usually complete and absolute, and is associated with severe colicky pains, vomiting and symptoms of collapse. But in acute intussusception there is oozing of blood mixed with mucus from the anus. In chronic obstruction the condition of the bowels varies from time to time; thus when the obstruction is due to cancer of the rectum or sigmoid flexure, diarrhœa and constipation may alternate, but in obstruction from fæcal accumulation, constipation is pronounced, and tends to become absolute.

In order to differentiate the various forms of intestinal obstruction, it is necessary to make a careful examination of the abdomen and its parietes, to consider the sex, age and previous history of the patient, and to study the mode of onset and the character of the pain, vomiting, and other symptoms that may be present. These points will be found discussed in text-books on medicine or surgery.

A digital examination of the rectum should be made when pain is referred to the rectum or to the lower part of the abdomen. Pain and straining at stool (*tenesmus*) occur in dysentery and other inflammatory affections of the descending colon, also in connection with piles, fistula, stricture, or other lesions affecting the rectum.

The **rectal crises**, which sometimes occur in locomotor ataxia, are characterised by paroxysms of severe pain and tenesmus, and the patient may complain that he feels as if there were a foreign body in the rectum which he has a strong desire to evacuate.

IV. SYMPTOMS INDICATING DISTURBANCE OF THE URINARY ORGANS.

The symptoms to which a patient may call attention are:—Changes in the appearance or quantity of urine (see Examination of Urine, Chap. X.); alterations in the frequency of micturition; inability to pass water or to hold it properly; pain or difficulty in micturition.

Increased Frequency of Micturition must be distinguished from alterations in the quantity of urine passed. Thus in stricture of the urethra, prostatic disease, cystitis, tumour or calculus of the bladder or

to empty itself properly. It is therefore always partially and often completely filled; hence urine is discharged frequently, and often "incontinently." The association of incontinence with retention occurs also in females as a result of prolonged labour, and in girls from vulvitis or other variety of genital irritation. Retention with "overflow incontinence" is also an important symptom in many cases of locomotor ataxia and other diseases of the spinal cord. It is common, too, in conditions of mental impairment from whatever cause. True incontinence of urine from paralysis of the sphincter is produced by lesions of the cord which implicate the bladder centre in the lumbar enlargement. Wetting the bed or the clothes is sometimes a valuable indication of the occurrence of an epileptic fit.

Nocturnal incontinence of urine apart from epilepsy or any local lesion is not uncommon in children; the urine is usually voided during the early hours of sleep. The symptom is to be regarded as a sign of nerve weakness, of undue tendency to reflex action. Sometimes the affected child exhibits rheumatic proclivities; but careful search should always be made for any sign of local irritation, such as phimosis, worms, or vulvitis.

Pain, with or without Difficulty, in Micturition, occurs in a number of morbid conditions affecting the urethra or bladder. It is common in pelvic inflammation, especially in women, and in cases of enlargement or displacement of the uterus. Pain and a frequent desire to pass water are prominent symptoms in cystitis, and in cases of stone in the bladder. The pain in calculus is usually referred to the end of the penis, and is much increased by sudden movements of the body. In cystitis, pain is situated over the pubis and sacrum, and in the perineum; there is also a very urgent desire to pass water, which is experienced even after all the urine has been voided.

Pain referred to the bladder and frequent painful micturition requires especial emphasis in relation to peritonitis which has spread down to Douglas's pouch. This is common in disease of the vermiform appendix and in latent peritonitis from typhoid fever. Here, too, may be mentioned the **nephralgic, vesical, and urethral crises**, which occasionally manifest themselves during the course of locomotor ataxia. In nephralgic crises the paroxysmal pains closely resemble attacks of renal colic, while in bladder and urethral crises the patient suffers from painful and frequent micturition, which very rarely is accompanied by the passing of a little blood.

CHAPTER III.

EXAMINATION OF THE SURFACE OF THE BODY.

UNDER this heading may be included the examination of the skin and its appendages, that of the subcutaneous tissues, of the contour of the muscles, glands, bones and joints, together with a brief notice of tumours and deformities.

The deviations from normal are extremely numerous, and it is only possible, with the space at our disposal, to give a brief account of the more important ones; of these many will be dealt with in the chapters devoted to diseases of the skin and of the nervous system, while others are referred to in the sections relating to inspection of the chest and abdomen.

In the present chapter the chief objective signs noticeable in making an examination (1) of the body generally, and (2) of the head, limbs and spine, are more particularly considered, and they may be conveniently arranged under the headings, Size, Shape and Expression. The term "expression" is taken to include not only the expression of the face, but also that of the body and limbs, as exemplified by posture, station and gait.

CHANGES IN SIZE AND SHAPE.

I. The Body Generally.—In a large number of diseases the general bulk or weight of the body becomes diminished; in comparatively few does it become increased.

When the whole body is wasted, we speak of emaciation or general atrophy; while wasting of a portion of the body is called local atrophy. In general atrophy the most obvious sign is wasting of the subcutaneous fat. In progressive muscular atrophy, which may also produce great thinness of limb and body, there is not, strictly speaking, general atrophy, but general atrophy of one tissue, namely, the muscular. Local atrophy commonly depends on loss of muscular tissue, but the bones and other tissues may also be involved.

In muscular atrophy the strength of individual movements is impaired, and the rapidity with which the weakness progresses varies with the position and nature of the lesion; but in cases of adipose atrophy, voluntary power is not necessarily affected. At the same

time the two varieties cannot be abruptly separated, for whenever wasting of adipose tissue occurs as a result of disease, the muscles also suffer, if only to a slight extent.

Emaciation (chiefly Adipose Atrophy) is roughly estimated by the ease with which a fold of skin is pinched up from the underlying parts; but in order to obtain accurate indications the scales are necessary, and in all serious diseases the patient should be weighed, if practicable, at regular intervals, for then useful information is obtained as to the progress of the malady.

Emaciation is a prominent feature in all acute febrile diseases, and is also present in varying degree in the majority of chronic maladies. It is especially noticeable in typhoid fever, where it progresses with greater rapidity than can be accounted for by the degree of pyrexia, the scanty diet, or the loss by the evacuations. Emaciation is frequently one of the earliest indications of phthisis, also of tubercular meningitis in children, in whom it may occur for some time before the onset of more characteristic symptoms.

In pulmonary phthisis or in general tuberculosis the chest and limbs are the most affected, while the face is often spared till the malady is far advanced; but in malignant disease, and especially when the abdominal organs are involved, the face wastes as much and as rapidly as other parts of the body. In infancy, unsuitable food or catarrh of the stomach and bowels are the commonest causes of general atrophy. It is sometimes present in rickets and congenital syphilis, but in both these diseases the infant may be plump and well nourished, and indeed "fat rickets" is commoner than "lean rickets." Occasionally cases of infantile atrophy are met with to which no clue can be found: a proper quantity of suitable food is taken, the digestive and other functions appear to be normal, there is no pyrexia, and no lesion is discovered on post-mortem examination. It should be observed that in children weight is more readily lost, more quickly regained, and that its loss is less frequently a sign of serious disease than in adults. In the latter, however, and especially in females, extreme emaciation may occur in association with hysteria, but this no doubt is mainly the result of fasting; and in all cases of emaciation the question of starvation, whether voluntary or enforced by necessity or disease, must be carefully considered.

Local Atrophy.—In local atrophy the skin, fat, muscles and bones may be separately or collectively involved. Thus atrophy of the skin may result from undue stretching of a part, witness the "lineæ albicantes" on the abdomen of women as a result of pregnancy, or on the breasts as a result of lactation.

Atrophy of the hand muscles may be produced by destructive lesions

of the median and ulnar nerves; progressive wasting of a limb from destruction of cells in the anterior horns of the spinal cord. In the latter condition the bones are affected as well as the muscles, but the wasting of the osseous tissue bears no proportion to that of the muscular, for the bones may be found of normal length and thickness when the greater part of the muscles of a limb is lost, and conversely a limb may be shortened and thinned when its muscles are free from obvious wasting. The condition of a limb in which a retrograde change has taken place in parts originally well developed must be distinguished from a limb in which there has been **arrest of growth.** Thus a destructive lesion of the motor part of the cortex on one side of the brain in early infancy will hinder the growth of the limbs on the opposite side of the body, and hence at a later period of life they will be shorter and thinner than their fellows, the bones as well as the soft tissues being more or less arrested in their development. The affected limbs, indeed, in some cases may be perfect in shape and correspond to those of a healthy child, while the opposite limbs have the size and vigour of adult life.

The presence or absence of local muscular atrophy is determined first by inspection and palpation, a muscular prominence on one side being compared as to bulk and consistence with the same part on the other side of the body; second, by measurement; third, by testing the strength of the part supposed to be affected.

The conditions under which atrophy of muscle occurs may be arranged in four classes:—*Atrophy from disuse:* thus a moderate degree of wasting affects the muscles of a paralysed limb, or of a limb that has been kept in splints for a length of time. *Atrophy from disease of the muscular tissue itself:* the so-called "myopathic atrophy." *Atrophy from disease of the nervous system.* Examples: infantile paralysis, the muscular atrophy depending on destruction of the cells in the anterior horns of the cord; wasting of the extensor muscles of the forearm from disease or injury of the musculo-spiral nerve. *Arthritic Atrophy:*—When a joint is inflamed, either from injury or disease, the muscles moving it frequently undergo rapid wasting: thus wasting of the thigh muscles follows severe injury to the knee-joint; atrophy of the interosseous muscles of the hands attends rheumatic swellings of the knuckles. The distinctions between these varieties of muscular atrophy are given in the section on the Nervous System.

Obesity, or an excessive quantity of fat throughout the body, is of frequent occurrence in women at or about the climacteric period; also in persons who habitually indulge too freely in malt liquors. It is common, too, in idiots, and in some cases of chronic cerebral disease, *e.g.*, cerebral tumour.

By **Dropsy** is meant an accumulation of serous fluid in the areolar spaces of the connective tissue, or in the serous cavities of the body.

In the former position, dropsy, when extensive, is termed **Anasarca**; when more or less localised, **Œdema**. Dropsy increases the size of the affected part, and the swelling is distinguished by the pit which is produced on pressing with the finger. The degree of enlargement varies much: it may be so great as to render the skin tense and shining, or so slight as to be scarcely perceptible. In doubtful cases, as, for example, when examining the leg, steady continuous pressure with the finger over the shin for a few seconds may produce slight pitting when more sudden pressure has been unsuccessful; also even in minor degrees of œdema, a feeling of doughiness or want of elasticity is usually experienced. As a rule, the pit begins to gradually fill up directly the finger is removed, but in extreme œdema it may persist for a considerable time.

Dropsy is most marked in dependent parts, and in regions where there is much loose cellular tissue, as the eyelids or scrotum; it should always be looked for over the sacrum, even when undetectable elsewhere.

Dropsy may result from any cause which interferes with the circulation of the blood, or which leads to deterioration of this fluid itself.

Œdema **limited to the legs**, slight or moderate in degree, occurring in middle life, and especially in females, is frequently due to varicose veins; in such cases, even if there be no obvious varicosity of the superficial veins, the possibility that the deep veins may be affected should not be forgotten. Œdema of the lower limbs may also be caused by any undue pressure within the abdomen, as from tumours or ascites. Œdema **beginning about the ankles**, and then slowly progressing to other parts of the body, occurs usually as a result of mitral regurgitation or of mitral stenosis; it is also produced by dilatation of the right side of the heart, due either to obstruction to the pulmonary circulation, as from emphysema and bronchitis, or to weakness of the cardiac muscle. Slight puffiness about the ankles, becoming a genuine œdema **after standing or walking**, is significant of anæmia; and the amount of dropsy, originally started by mechanical obstruction to the circulation, is largely influenced by the degree of anæmia present.

Puffiness **beneath the eyes** may be noticed in whooping-cough, and is an early sign of renal dropsy. In acute nephritis the spread of œdema is often very rapid; in a few hours the features may be almost obliterated and the whole body greatly swollen; the subsidence of the swelling may be equally rapid.

In some forms of chronic Bright's disease there is considerable and persistent anasarca, but in the red granular kidney, œdema may be absent or quite insignificant. Apart from kidney disease, œdema of

the legs and considerable puffiness of the backs of the hands is not uncommon in marasmic infants.

Dropsy **limited to the arms and upper part of the body** points to

FIG. 1.—Face of Large White Kidney (Dr. Dreschfeld's Case). The left eye is nearly closed by œdematous swelling. The raised eyebrows and wrinkled forehead indicate the difficulty in opening the eyes.

mechanical obstruction within the thorax, as from a mediastinal tumour pressing on the superior vena cava.

Dropsy **limited to one limb** indicates obstruction of a venous trunk, as, for example, the swollen lower limb of phlegmasia dolens. Local

FIG. 2.—Face of Woman, aged 45, showing moderate degree of Myxœdema.

œdema may also result from active congestion; thus the inflammatory swelling of gout pits on pressure.

Myxœdema.—In this disease there is an increase in the general

bulk of the body, the whole surface is swollen, but does not pit on pressure, and the skin tends to be dry and scaly. The face becomes broad, puffy, and expressionless, and glistening pear-shaped swellings are sometimes to be seen below the eyes. The nostrils are swollen and the lower lip is thickened and everted. The hands and feet are large

FIG. 3.—Dr. Dreschfeld's Case of Acromegaly. Duration of the disease five years. The enlargement of the lower jaw and of the hands and feet were conspicuous features. The thyroid could not be felt, but on each side of the neck there was a well-defined swelling, which dipped down into the thorax, and was continuous with an area of dulness over the upper part of the sternum.—*B. M. J.*, January 1894.

and spade-like. The tongue too is enlarged. There is usually torpor of both mind and body, together with some impairment of the special senses, and the utterance is thick, slow, and guttural. The temperature of the body is usually subnormal and the urine contains a deficient quantity of urea.

Acromegaly, a rare disease, resembles myxœdema in many respects, but presents many notable differences. Thus the skin is but rarely hard and dry, and the bodily and mental functions are not impaired. Also the hands and feet are greatly hypertrophied; the face, too, is elongated, and certain parts, especially the nose and lower jaw, become much increased in size. Frequently the ends of the long bones are hypertrophied, but the shafts are unaffected. Sometimes temporal hemianopsia is present. Mediastinal dulness in consequence of enlargement of the thymus gland has also been detected.

Subcutaneous Emphysema, a condition caused by the escape of air or gas into the subcutaneous tissue, produces a swelling somewhat similar in appearance to that of œdema. On pressure with the finger, a characteristic feeling of crackling is experienced; a pit is also produced, which fills up more quickly than that of œdema. Variable and, as a rule, limited in its distribution, emphysema may extend over nearly the whole body. Apart from surgical cases, it is usually set up:—(1.) By rupture of some of the pulmonary air cells in consequence of great intra-thoracic pressure, as in whooping-cough; the air is driven into the inter-lobular septa, and then finds its way through the mediastinum into the cellular tissue of the neck. (2.) By ulceration over a pulmonary cavity, which has become adherent to the chest wall. (3.) By perforation from ulcer of the larynx, trachea, œsophagus, stomach, or intestine. In the case of the stomach and intestine, it is necessary for the affected part to be glued to the abdominal wall, otherwise the gas will escape into the peritoneal cavity.

Local increase in size, other than that caused by obesity, dropsy, subcutaneous emphysema, the presence of a new growth, aneurysm, or other tumour, may be caused by true or false hypertrophy of the muscular tissue. *True hypertrophy* of muscle, as a result of disease, is exceedingly rare. It occurs in Thomsen's disease, the characteristic symptom of which is rigidity of muscles when put into action after a period of rest. Here the hypertrophy is often accompanied by increased strength; but in a still rarer affection a partial or wide-spread hypertrophy of muscle is found in association with diminished power.

False Hypertrophy of muscle is a leading feature in the disease known as pseudo-hypertrophic paralysis, where the increased size of the calves and other muscular masses is due to a growth of fatty or fibrous interstitial tissue.

Enlarged hard muscles also occur in cretinism, and may supervene on muscular atrophy, as, for example, sometimes in infantile paralysis and in hemiplegia.

II. The Head and Face.—Variations in size and shape, within the limits of health, and apart from mental impairment, are innumerable,

and will be found discussed in works on Anthropology. Variations depending on pathological changes are chiefly met with in early life.

The cranium of even the youngest healthy infant is firm and unyielding to ordinary pressure, except over the anterior and posterior fontanelles; the two halves are almost, if not quite, symmetrical; the shape of the head in the antero-posterior plane is that of an irregular pentagon with curved sides, and this form is maintained throughout life. The size is variable, but the following may be taken as normal limits below the age of five years:—The circumference at the level of the occipital protuberance, from 15 to 20 inches; the transverse diameter,

FIG. 4.—Child the subject of Rickets. Head shows bossy frontal eminences. Thorax shows anterior convexity (sternum and costal cartilages), lateral grooves, and transverse constriction. Abdomen large.

taken with the calipers between the parietal eminences, from 4 to 6 inches; and the antero-posterior diameter from 6 to 8 inches. Ample illustration of changes in the form of the head and face will be afforded by a brief reference to rickets, cretinism and a few other familiar diseases.

Rickets.—In infants a few months old the free margins of the flat bones of the skull may be unduly soft, and the occiput and the parietals may yield to the pressure of the finger like parchment; also round spots of local thinning may be detected on these bones, and even exceptionally

on the frontal. To this abnormal flexibility the term **Craniotabes** is applied. Associated with craniotabes, bossy swellings may form on the frontal and parietal bones in front of and behind the anterior fontanelle respectively; they shine through the thin pale scalp, and sometimes cause a characteristic pale bluish prominence. There is no local heat or tenderness over these areas, but it is probable that the irritability displayed by infants so affected, the throwing about of the head and the boring into the pillow, bear a direct relation to these hyperplastic changes. In aggravated cases the bosses increase in size, and other swellings arise around the parietal eminences, on the upper part of the occiput, on the temporal regions and in the neighbourhood of the sutures. All these new superposed osseous growths, if not absorbed, gradually become more or less diffused and organised, and thus give rise to the various forms of the rickety skull. Of these there are two principal shapes to be noticed: the commonest shape presents a broad, square forehead, strongly-developed frontal and parietal eminences and occipital protuberance; the crown is flattened, but still shows some indications of the original four bosses, with a broad median groove and a ridge on each side of it. The second type of rickety skull is elongated fore and aft, or markedly dolichocephalic.

Other rickety heads show the whole occipital region flattened so as to appear nearly vertical on side view; some show marked asymmetry, especially in the posterior part, and this is occasionally accompanied by compensation in the fore part; the frontal region being prominent on the same side as the flattened parieto-occipital region, and *vice versa*. Delay in closure of fontanelles and sutures is also to be noticed. The anterior fontanelle, which in a healthy child is usually closed at about eighteen months of age, may be unduly wide long after this period. Grooves may also be felt in the cranial bones for the distended veins which course over the scalp. The face in rickets often looks small in contrast to the massive frontal region; the alveolar border of the upper jaw tends to assume a beak-like shape, while the lower jaw is somewhat polygonal, with its anterior part turned slightly inwards.

Cretinism.—In this disease the head is large, and in many cases brachycephalic—that is, contracted from before back and expanded at the sides—and sometimes measures more from ear to ear than from the root of the nose to the occipital spine. The top and back of the head are usually flattened. The face is square and large, especially in the upper third; the nose is short, depressed at its root, and spreads out enormously towards the alæ; the eyes are wide apart, the mouth large and gaping, and the lips thick.

Enlargement and thickening of the bones of the cranium may also be due to injury, syphilis, ostitis deformans, or leontiasis ossea.

In **Hereditary Syphilis**, after the period of infancy, the forehead is often square and upright, and is prominent at and within the frontal eminences; a somewhat characteristic feature of the prominence is a

FIG. 5.—Dr. Shuttleworth's Case of Cretinism. "Sarah," aged 21. Height, nearly 3 feet; weight, 49 lbs. Features characteristic. Frontal suture and anterior fontanelle not completely closed. Speech slow, and limited to a few words.

ridge placed transversely between the frontal eminences. But this is not constant, and skull changes due to syphilis are apt to be associated with skull changes due to rickets. Occasionally necrosis and exfoliation of bone take place, and may leave a large gap in the frontal or parietal region. In young infants asymmetry of the skull is sometimes present.

In association with the above phenomena characteristic changes may be observed in the face. The corneæ present opacities, the bridge of

FIG. 5.—Child the subject of late Hereditary Syphilis.

FIG. 7.—Chronic Hydrocephalus in a young child.

the nose is thickened and depressed, and radiating linear scars are to be seen about the nose and mouth.

Hydrocephalus.—The pentagonal shape of health tends to become a

circle. The head is round, increased in height out of proportion to its length, and usually bulges more in front than behind. The circumference may measure as much as thirty inches. The fontanelles and sutures are widened out and unduly prominent, and frequently fluctuation can be obtained. The cranial bones become very thin, either

Fig. 1. *

Fig. 2.

Skull and Brain from Case Fig. 1. The skull shows large fontanelle and numerous infantile spots. The brain shows great enlargement of the lateral and fourth ventricles.

generally, when sometimes a crackling sensation may be yielded on pressure; or in limited areas, exostoses. In comparison with the big cranium the lower part of the face looks abnormally small. The eyeballs are prominent, and are depressed so that the sclerotic above

the cornea is often exposed while the pupils may be partly covered by the lower lids.

In young children, especially during the latter half of the first year, it is often difficult to decide whether hydrocephalus is present or not.

FIG. 10.—Boy with small conical head, internal squint, defective intelligence, and spastic limbs. Almost daily convulsions from age of six months till death at age of two years.

The child's head enlarges quickly, the fontanelle is full, and perhaps a fit occurs; in such a case it is useful to compare cyrtometrical tracings made at monthly intervals.

FIG. 11.—Brain from Case Fig. 10, shows a deep sulcus behind the frontal lobe, and imperfectly formed convolutions behind the sulcus. The cerebellum was uncovered as in Fig.

Microcephalus.—The head may be smaller than natural and yet maintain a normal shape; in some cases, however, the diminution in

size is due chiefly to great narrowing of the transverse diameter of the frontal region, while the antero-posterior plane remains of normal length; in other cases the head tapers towards the top and presents a triangular shape in the coronal section. The sutures and fontanelles are prematurely closed.

A microcephalic child is more or less idiotic, is subject to fits, and frequently has rigid flexed limbs (Fig. 10).

Asymmetry of Skull has already been mentioned as occasionally present in rickety and in syphilitic infants. It also occurs in association with defects in one of the cerebral hemispheres, when there may be a condition of crossed atrophy. Thus if some of the convolutions on the left side of the brain are wasted or wanting, the left parietal bone may feel flatter than the right, and the left side of the forehead shore

FIG. 12.—Girl, aged 9. Right limbs, right half of chest, and right cheek smaller than corresponding parts on left side. Marked atrophy of right half of *tongue*. Above the cheek, however, the *left* side of the head was a little smaller than the right side.

off more than the right side; but below the eyes the arrested growth will affect the right cheek and the right limbs, the latter frequently presenting a spastic hemiplegia. Slighter degrees of this asymmetry are by no means uncommon; indeed, it is perhaps rare to see perfect equality between the two sides of the face, and it may be that there is a corresponding asymmetry between the cerebral motor areas. A still rarer form of facial asymmetry is seen in the affection known as "**unilateral atrophy of the face**;" in this disease the skin, connective tissue, and fat are thinned and wasted whilst the muscles are spared; the bones too, if the atrophy begins in early life, may be arrested in their development, and then the two halves of the face look as if they belonged to different individuals; one side having the fulness of youth, the other the wrinkles of old age (Fig. 13).

the thoracic aorta or other mediastinal tumour, and in injury or disease of the cervical portion of the spinal cord, and a diagnosis between these conditions may often be instantly made by a general inspection of the body; for in the case of aneurysm we should probably at once notice some pulsation of the upper part of the chest wall; and in affections of the cervical cord, muscular atrophy in the upper limbs and the position of the hands and the spastic attitude of the feet would attract our attention; while in locomotor ataxy there would be an absence of all these signs, but both pupils, besides being small, would be inactive to light. They are also diminished in size in mitral regurgitation, in typhus fever, and whenever the iris is congested either from general or local causes. Great contraction of the pupils, in association with profound coma, if not due to opium poisoning, suggests hæmorrhage into the pons Varolii.

Dilatation of the Pupils (Mydriasis).—The pupils are larger in childhood than in adult life. They are often dilated in hysteria, in anæmia, in typhoid fever, and in many cases of apoplexy.

Inequality of the Pupils may be due to unequal refraction of the two eyes, myopia being associated with mydriasis, hypermetropia with myosis. A destructive lesion of one third nerve produces dilatation of the corresponding pupil. Inequality of the pupils is often a prominent symptom in general paralysis of the insane; it occurs sometimes in locomotor ataxy, also in migraine.

The effect of poisons on the size of the pupil must be remembered —atropine, duboisin, and cocaine dilate, while opium, eserine, and pilocarpine contract the pupil.

Irregularities in Shape suggest iritis; they are also frequently met with as a result of iridectomy. Coloboma of the iris is a congenital cleft in the iris, which is always directed downwards or slightly down and in. It may affect one or both eyes, and may occur with or without coloboma of the choroid.

IV. The Limbs.—In addition to wasting or overgrowth of the soft tissues of a limb, which have been already referred to, its size and shape may be considerably altered in consequence of disease of the bones or joints; and the following are some of the more important deformities which result from such changes.

In **Rickets** the earliest naked-eye changes in the upper limbs are observed at the wrists, the lower ends of the radius and ulna being larger than normal. This enlargement is usually most obvious between three months and two years of age, a period when beads at the junction of the ribs with their costal cartilages are also prominent. The lower ends of the tibiæ, and, to a less degree, the ends of the other long bones, also show enlargement.

The chief changes in the shafts of the long bones are as follows:—The arms become convex outwards about the insertion of the deltoid; the forearms convex backwards; the thighs convex forwards, and sometimes outwards as well; while the tibiæ present a slight concavity on the inner surface, or a marked forward convexity in their lower thirds.

Mollities ossium, a rare disease, chiefly affecting the female sex, is characterised by fractures and extreme flexibility of the long bones, together with distortions of the spine, sternum and pelvis.

In **Cretinism** the ends of the bones are frequently abnormally large; the hands are spade-like, and the fingers and toes shorter than normal. (See Fig. 5.)

In infants of about three months old who are the subjects of **Inherited Syphilis**, slight swelling may occur in the neighbourhood of the wrists, elbows, shoulders, or knees, together with a "pseudo-paralysis" of these parts. Tenderness and powerlessness are usually more marked than swelling. The enlargement at the wrist is just above the junction of the epiphysis of the radius with its shaft, and is therefore a little higher than that of rickets; it may also extend for a short distance along the shaft. Occasionally suppuration or partial dislocation of the epiphysis occurs, which subsequently becomes welded with some displacement to the shaft.

Older children, just as the subjects of acquired syphilis, may present nodose or diffuse enlargement of the bones, especially the ulnæ, lower ends of humeri, clavicles and tibiæ; and there may be overgrowth in length as well as in thickness.

Nodose bony swellings occasionally develop during the convalescent period of **Typhoid Fever**. They are usually very painful to pressure, and are accompanied by pyrexia, and some spontaneous pain in the affected limb.

In **Infantile Scurvy**, at from sixteen to eighteen months of age, there may be noticed extreme tenderness, swelling and immobility of the lower limbs, and sometimes of the upper limbs. The affected part, commonly the thigh, is swollen and cylindrical in shape, tense and shining. The swelling, which is deep-seated, begins near the junction of shaft with epiphysis, and extends for a varying distance along the shaft. The child is somewhat wasted, pale, sallow and fretful. Its gums are frequently spongy, and liable to bleed. The urine may contain albumen or blood.

Thickening and Spontaneous Fractures of Bones are occasionally produced by injuries or diseases of the nervous system. Thus injuries of nerve trunks have been followed by swelling and thickening of the bones; and if the injury occur in early life, the development of the affected bones may be arrested. Deformities and fractures of the

bones occur in the insane, and particularly in general paralytics; also in locomotor ataxy, in which disease the period of swelling and fracture is usually preceded by paroxysms of lancinating pains.

Very rarely spontaneous fractures occur in young infants, and probably as a result of rickets. They occur in the middle of the shaft, as well as near the epiphysis; they may be single or multiple, partial or complete.

Clubbing of Fingers and Toes.—By this is meant an enlargement of the ungual phalanges of the fingers or toes. Clubbing may be regarded as an indication of impeded circulation within the thorax. Its commonest causes are phthisis, empyema, and congenital heart disease. The thickening in phthisis is mainly from before back; in empyema, from side to side. In phthisis the nails are often filbert shaped and incurved. Incurvation of the nails—that is, a marked turning down of the tip of the nail—is occasionally met with in healthy persons, but, as a rule, the normal direction of the nail is a gradual upward slant from matrix to tip.

The Joints.—In medical practice articular swellings are most commonly due to rheumatism, gout, or rheumatoid arthritis. They also occur in connection with gonorrhea, syphilis, pyæmia, the puerperal state, some of the acute specifics, especially scarlet fever, purpura hæmorrhagica, hæmophilia, and during the course of certain diseases of the nervous system.

In **Acute or Subacute Rheumatism**, while any joint may be attacked, it is the larger and medium-sized ones which are principally affected. There is some tenderness and usually great pain on movement. The skin over the joint is natural in appearance or presents a pinkish blush. The arthritis is transitory and shifting in character and symmetry is usually displayed in the order of succession. Thus while the swelling of one knee is subsiding, the other knee is the most likely joint to be next attacked.

Pyrexia and profuse acid sweats accompany the arthritis, and appear to vary in severity with its intensity and extent. At all ages rheumatism exhibits migratory and relapsing tendencies, but in other respects certain differences are to be noticed at different ages. Thus rheumatism in the child is characterised by the frequency with which erythematous eruptions, subcutaneous nodules, endocarditis, pericarditis and chorea occur; while sweating, articular pain and swelling, and pyrexia are more marked in the adult.

With regard to the **nodules**, they are small firm bodies, varying in size from a pin's head to an almond, which are situated over the tendon sheaths or fasciæ, and also over bony prominences, and are most commonly found about the elbows, and over the extensor tendons of

the hands. Other sites are the margins of the patella, the malleoli, the vertebral spines, the parietal and occipital bones, and the bony prominences of the shoulders and hips.

FIG. 14.—Subcutaneous Rheumatic Nodules on the elbow of a lad aged sixteen. (*After A. Garrod.*)

Rheumatoid Arthritis may follow an attack of rheumatism, gout, or gonorrheal rheumatism; or be developed independently of any previous

FIG. 15.—Chronic Polyarticular Rheumatoid Arthritis.

disease. Three more or less distinct types can be distinguished, which, however, are not very rarely found in association.

The most important variety is *polyarticular rheumatoid arthritis*. It occurs chiefly in women about the climacteric period, but is also met with in both sexes, and at the two extremes of life. In the child it is rare and assumes a rapidly progressive form, while in advanced life it is usually extremely chronic. The small joints of the hands and feet are the first to be attacked, then the disease steadily progresses towards the trunk; in the upper limbs, for example, involving in order of succession the wrists, elbows and shoulders. Great symmetry is exhibited in the distribution of the lesions.

The temporo-maxillary articulation, rarely affected in acute rheumatism, is often involved in rheumatoid arthritis. The neck frequently becomes stiff in consequence of "spondylitis deformans" of the cervical spine. With regard to the hands, all the phalangeal joints may be enlarged, the enlargement being partly due to thickening of the articular ends of the bones, and partly to an increase of fluid in the synovial membrane. When the elbow is involved, the bursa over the olecranon is frequently distended with fluid. In advanced cases movements of the bony surfaces against one another produce a characteristic grating. The skin over the enlarged joints is sometimes glossy and pigmented, while the nails are brittle and longitudinally ridged. Atrophy of the neighbouring muscles is a striking feature, but visceral complications are rare.

Heberden's Nodes.—These are due to an osteophytic enlargement of the ends of the bones which enter into the formation of the terminal phalangeal joints. This variety often occurs alone, but also in association with the polyarticular variety.

The *Monarticular Variety* affects men oftener than women. The joint, usually the hip or shoulder, becomes stiff and painful, and the neighbouring muscles undergo marked atrophy.

It may here be mentioned that enlargement of the ends of the bones, a distinctive feature of the diseased joints in rheumatoid arthritis, is also occasionally present in acute or subacute rheumatism; for example, the author has observed thickening of the styloid and coronoid processes of the ulna, of the ends of the radius, and in one case, of the ribs near their anterior ends. There is sometimes, too, great tenderness over the thickened portion of bone, suggesting periostitis, and sometimes a fibrous nodule may be felt adhering to the presumably inflamed periosteum.

Gout.—The gouty joint, which is usually one of the small articulations, and most commonly the metacarpo-phalangeal joint of the great toe, is the seat of great pain and tenderness; the part is red, swollen and œdematous, and the skin desquamates towards the end of the attack. In chronic cases tophi or chalk stones may be found around

the joints and in the cartilages of the ears. The creamy juice obtained by pricking one of these deposits, when placed on a glass slide with a drop of liq. potassæ, shows under the microscope innumerable delicate needle-shaped crystals, which are chiefly composed of biurate of sodium.

When many joints are affected, and no tophi can be discovered, there is often a difficulty in deciding between rheumatoid arthritis and gout. The former often attacks more joints, and shows a greater tendency to symmetry than the latter disease. The history of the case is also of help in forming a diagnosis.

Gonorrhœal Arthritis.—This name is given to joint lesions which sometimes occur in connection with a purulent discharge from a mucous membrane, especially that of the urethra. The knees and feet are most commonly attacked. The feet become swollen, and the soles are often excessively tender. The temporo-maxillary joints are liable to be affected. The arthritis is of an intractable character; it may be progressive, and result in ankylosis of many joints, including spondylitis deformans. But, as a rule, the disease is limited to a few joints; while constitutional disturbance is slight. Conjunctivitis and iritis are not uncommon, but endocarditis is rare, and, when present, is nearly always of the ulcerative or septic variety.

In **Scarlet Fever** two types of joint affection may be distinguished. One variety occurs towards the end of the first week, and is usually transitory. The sheaths of the tendons at the back of the wrists, as well as the wrists themselves and other joints, become tender, red and swollen. The other variety comes on during the period of desquamation, and is frequently indistinguishable from ordinary rheumatism.

In **Hæmophilia** the knees sometimes become swollen in consequence of hæmorrhage into the joint, or from an effusion of serum into the tissues around it.

In **Congenital Syphilis** a passive synovitis is not uncommon as one of the later manifestations. A child walks into the out-patient room, and, on examination, one of the knees is enlarged, perhaps distended with fluid, but pain and tenderness are usually absent.

Diseases of the Nervous System.—Painful swelling of joints sometimes follows injuries or diseases of the spinal cord or peripheral nerves; also redness and swelling of the larger joints may be found on the paralysed side in hemiplegia, and oftener in cases of softening from thrombosis than in cases of hæmorrhage.

A form of chronic arthritis sometimes develops very suddenly in locomotor ataxy; the joint, usually the knee, hip, shoulder, or elbow, may be considerably swollen within twenty-four hours from the com-

commencement, and generally without pain or febrile reaction. Sometimes the joint recovers completely, but in severe cases the heads of the bones gradually become atrophied, and then, owing to relaxation of the ligaments and feebleness of the surrounding muscles, spontaneous luxations frequently occur. The joints of the foot are sometimes attacked and a characteristic deformity—the "tabetic foot"—is produced.

V. The Spine.—The various antero-posterior curves of the healthy spine are less marked in childhood than at a later period of life.

Fig. 16.—Extreme backward dislocation of left knee in a man aged 50, the subject of locomotor ataxy. There was also dislocation of head of right femur on to dorsum of ilium. (Dr. Dreschfeld's Case, *Lancet*, ii., 1880.)

Disease may cause an increase or diminution of the natural curvatures, a lateral curvature, or projections in certain parts of the vertebral column.

For example, an **increase in the dorsal curve** is observed in rickets when the child is placed in the sitting posture, the dorsal spines projecting backward to form a gradual rounded convexity; and if a child suffering from moderately severe rickets is often kept in the sitting posture, a permanent deformity may ensue, varying from a gradual curve up to a rounded gibbosity, and accompanied by some degree of lateral modification.

Diminution of the Natural Curves—that is, undue straightness of the spine combined with rigidity of the back muscles—is significant of spinal caries, even in the absence of any angular projection.

Lateral Curvature or Scoliosis.—In this distortion there are two or more lateral curves, and the vertebræ are rotated on their vertical axis, so that the spinous processes are deflected towards the concavity of the curve. On the convex side the scapula is projected backwards by the increased arching of the ribs—hence the popular name for the affection, "growing out of the shoulder"—while on the opposite side the breast may frequently be noticed to be unduly prominent.

The commonest causes in young children are rickets, pleurisy, and infantile paralysis affecting the muscles of the back. Other causes at all ages are:—(1.) Obliquity of the pelvis as a result of some abnormality of the lower limb, such as a natural inequality in the length of the legs, hip disease, or sciatica; (2.) Contraction of one side of the chest after empyema; (3.) Unilateral contraction or paralysis of the spinal muscles.

Projections.—A slight prominence of one or two spinous processes occurs sometimes in thin nervous subjects in association with backache and spinal tenderness. But a distinct **angular projection** points to disease of the bones with loss of substance; it may involve two or more of the vertebral spines, and is usually most evident in the dorsal region. Rigidity of the spinal muscles is a marked feature of such cases; pain and tenderness are less constant symptoms. **Irregularities** in the spinal column may also be the results of fracture or dislocation. When rheumatoid arthritis affects the spinal articulations, a prominence or local thickening may be present, together with limitation of movement, or complete fixation of the part. Occasionally the nerve roots become injured, which causes radiating pains, and a descending neuritis may be set up. In rare cases similar phenomena may be observed in syphilitic disease of the spine and membranes.

Spina bifida refers to a congenital malformation of the spinal canal, with protrusion of some of its contents in the form of a fluid tumour. This tumour is commonly situated in the lumbo-sacral region. Its cutaneous covering is usually thin and membrane-like, but occasionally is composed of normal skin. Associated with this malformation there are often clubfoot, paralysis of the lower limbs, and sometimes hydrocephalus. In cases of paralysis and anæsthesia of the lower limbs occurring in adults, it is important to examine the spine for the slight elastic swellings or depressions in the lower part of the vertebral column, which are sometimes present as indications of a "spina bifida occulta," which may have given rise to the paralytic phenomena.

VI. The Glands.—Enlargement of some of the lymphatic glands

is extremely common, and particularly so in early life; at this period any peripheral source of irritation, whether situated in skin or mucous membrane, and whether of simple or specific origin, is extremely liable to produce swelling of those glands which receive lymphatic vessels from the injured area. Thus the posterior cervical glands may be enlarged from a slight eczema of the scalp, or the glands below the lower jaw from a sore within the mouth. In seeking for an explanation of enlargement of any particular lymphatic glands, it is necessary to carefully examine the areas drained by them for some source of irritation. These areas and their chief lymphatic glands are arranged in the following table, after Ashby and Wright:—

DISTRIBUTION OF THE LYMPHATIC GLANDS AND THEIR DRAINAGE AREAS.

Glands.	
	Head and Neck.
Suboccipital Mastoid	Drain posterior half of head.
Parotid	Drain anterior half of head, orbits, nose, upper jaw, upper part of pharynx.
Submaxillary	Drain the lower gums, lower part of face, and front of mouth and tongue.
Supra-hyoid	Drain anterior part of tongue, chin, and lower lip.
Superficial cervical (lying beneath platysma)	Drain external ear, side of head, and neck and face.
Retro-pharyngeal	Drain nasal fossæ and pharynx (upper part).
Deep cervical—	
Upper set along carotid sheath.	Drain mouth, tonsils, palate, lower part of pharynx, larynx, posterior part of tongue, nasal fossæ, parotid and submaxillary glands, interior of skull, and deep parts of head and neck.
Lower set in supra-clavicular fossæ.	Drain upper set of lymph glands, lower part of neck, and join axillary and mediastinal glands.
	Upper Extremity.
Supra-condyloid	Drain three inner fingers.
Axillary	Drain upper extremity, dorsal and scapular regions, front and sides of trunk and breast.
	Lower Extremity.
Anterior tibial and popliteal.	Drain the deep lymphatics of the leg and receive some vessels from the skin of the leg and foot, chiefly the outer side.
Inguinal—	
Femoral set (superficial)	Drain superficial vessels of lower limb and partly of buttock and genitals, also perineum.
Horizontal set (superficial).	Drain abdomen below umbilicus, buttock and genitals. The deep vessels of the lower limb go to the deep glands along the femoral vein.
Iliac	Drain the pelvic viscera and the deep vessels of the genitals partly.

Glands.	*Lower Extremity.*
Lumbar	Drain all the lower glands, uterus, ovaries, testes, kidneys.
Sacral	Drain the rectum.

Roughly the umbilicus is the watershed draining to the axilla and groin, but the vessels cross and overlap both vertically and horizontally.

The term **Scrofulous Glands** is commonly applied to a slow painless enlargement of lymphatic glands, in which caseation is apt to occur early. Scrofulous glands sometimes develop in children who appear to be in perfect health, but they must, nevertheless, be regarded in all cases as a form of local tuberculosis, and therefore as owning a specific origin and having dangerous tendencies.

Lymphadenoma or **Hodgkin's Disease** is characterised by an enlargement of the lymphatic glands throughout the body. In the commonest cases the cervical glands are primarily involved, and they may form enormous hard masses on the sides of the neck, compressing in some cases the jugular vein or carotid artery. Results of mechanical pressure may also be witnessed in other parts of the body, as hydrothorax from pressure on the azygos or bronchial veins, cyanosis and dyspnœa from enlargement of the mediastinal glands, or ascites from enlarged glands pressing on the portal veins.

Frequently the spleen is enlarged, there is anæmia and proclivity to hæmorrhage; while pyrexia, intermittent in type, is usually a marked feature.

An important distinction between the glandular enlargements of Hodgkin's disease and those due to scrofula is that the former exhibit less liability than the latter to inflammatory extension and to fusion of glands by inflammation around them; hence the enlarged glands which compose the glandular masses of Hodgkin's disease remain discrete, and, as a rule, can be distinguished by palpation.

The Thyroid Gland.—It is not always an easy matter to feel this gland in health, but very often, especially in thin necks, it may be felt as a thin flattened mass of firm consistence on each side of the thyroid cartilage and upper portion of the trachea, while lying across the latter the isthmus may sometimes be distinguished. It is absent or diminished in size in myxœdema and in many cases of cretinism; also even when it is found enlarged in cretinism it is probable that the gland tissue itself is atrophied or destroyed in consequence of cystic or fibroid changes. In the disease known as exophthalmic goitre, Graves' disease, or Basedow's disease, enlargement of the thyroid gland is associated with prominence of the eyeballs and palpitation of the heart. The swelling is not usually very great or very hard, yet it may compress

the trachea to a dangerous degree; as a rule, it is more marked on the right than on the left side, and it is to be observed that, when the exophthalmos and thyroid enlargement are greatest on the right side, cardiac symptoms are usually severe. Other symptoms are profuse sweating, paroxysmal attacks of diarrhœa, occurring oftener at night than in the day; sometimes vomiting, anæmia and emaciation; while a fine rhythmical tremor of the limbs is almost a constant feature.

The Salivary Glands are larger in women than men; they gradually atrophy with old age. The parotid gland is subject to two forms of

FIG. 17.—Exophthalmic Goitre in a child aged 3½. All the symptoms present with the exception of tremors. (Dr. Agar Renshaw's and Dr. Dreschfeld's Case.)

enlargement. The *first* form is a simple enlargement, which is usually bilateral, but may be unilateral at first; it never suppurates. This enlargement is the prominent feature of mumps, and is attended by great pain and tenderness. In mumps the submaxillary glands are also sometimes affected, while during the subsidence of the swelling of the salivary glands, the testicle, ovary (?), or mamma may become tender and swollen; and at this period it occasionally happens that some of the joints of the lower limbs are painful and swollen. The *second* form is the suppurative parotid bubo, which is generally unilateral, and

resulting lines, seen on the face at rest, inform us of the presence of a certain state of mind, or of an alteration in the condition of the nervous system, or of the organs over which it presides.

Increased activity of the facial muscles leads to fixation of the features, either temporary or permanent, or to actual visible movements. Diminished activity of the facial muscles produces absence of expression.

Fixed Expressions.—The fixation of the features may be only of short duration, as when the result of a sudden attack of pain; or permanent lines and furrows may form owing to the constant overaction of certain muscles.

The expression of **pain** is a complex one; it is the result not only of pain *per se*, but of the effect on the particular tissue injured. Thus gastric pain is frequently indicated by a curved furrow which starts just above the wing of the nose, and makes a sharp curve outwards and downwards towards, but at some distance from, the angle of the mouth; when well developed it uncovers the canine tooth, and closely resembles the sneering or scornful expression.

These gastric lines are sometimes seen in typhoid fever, and at a late period of a severe case may be extremely marked, producing a typical sardonic grin. But the dominant and characteristic facial expression in typhoid is one of relaxation or loss of muscular tone, and the presence of risus sardonicus is usually an indication of severe abdominal mischief, such as deep ulceration or peritonitis.

When sudden pain is produced, as by pressure over the cæcum in a case of typhilitis, the retraction of the upper lip is accompanied by a descent of the diaphragm and fixation of the muscles of the abdominal wall: the movement is evidently one of protection.

In thoracic pain, as, for example, the sudden stabbing pain of pleurisy, a quick, shallow inspiration is taken by means of the intercostal muscles, the diaphragmatic movement being in abeyance, and the chest is temporarily fixed; the muscles of the face, too, participate in the upward movement; the mouth is slightly opened, the nostrils are dilated, and the eyebrows often raised. This opening out of all the features is also characteristic of the more chronic forms of thoracic distress; whereas, in the various affections of the abdominal organs there is a tendency to contraction of the features, the lips are often compressed, the angles of the mouth drawn down, and the face may express irritability, discontent, or profound depression, forming a marked contrast to the bright cheeks and sparkling, hopeful-looking eyes sometimes seen in advanced disease of the lungs.

An expression of marked **anxiety** is typically seen during a paroxysm of asthma, and is characteristic of the faces of many cases of cardiac

dyspnœa. The agony of **terror** is strikingly exhibited by sufferers from œdema of the glottis, or during an attack of angina pectoris. It is also, curiously enough, imitated, the subjective feeling of fear being absent, in cases of exophthalmic goitre, and in those rare cases of ocular nuclear paralysis where the superior recti are solely or mainly affected. Thus, in a patient of Dr. Morgan's (see Fig. 18), when she attempted to look upwards, the upper eyelids were retracted, but the eyeballs remained with their axes fixed in a downward direction, and this position, together with the wrinkled forehead, widened nostrils and half-open mouth, produced an expression which reminded one of the picture of terror in Charles Bell's book on the anatomy of expression.

FIG. 18.—Paralysis of Superior Recti Muscles.

A look of **astonishment** may also be permanently stamped on the countenance as a result of disease, and quite apart from any corresponding emotion experienced by the patient. Thus in cases of paralysis agitans, the eyebrows are raised, the forehead is marked by deep transverse wrinkles, and the mouth often remains open; the dull look of the eyes and the meaningless aspect of the lower part of the face render the imitation an imperfect one; yet in some cases the resemblance is striking. Also in that form of torticollis in which the head is jerked backwards, the forehead is transversely wrinkled, and the eyebrows are raised because of the physiological association between the frontales and the muscles that retract the head. The same expression is also temporarily imitated by a stupidly drunken person, who tries to overcome the drooping of the eyelids by raising his eyebrows and contracting his forehead, as is well represented in one of Hogarth's drawings.

Of other instances of altered expression in disease may be mentioned the frowning expression in some brain affections, the far-off fixation look in meningitis, the look of self-satisfaction in commencing paralysis of the insane, the malignant look in mania, the hopelessness in faces where there is melancholia and a disposition to suicide, the suspicious look of the alcoholic, and the mischievous face sometimes seen in a child who is subject to fits. Indeed in insanity and in hysteria every possible passion or emotion may be strikingly expressed.

Facial movements are frequently of diagnostic and prognostic

significance: thus twitching of some of the facial muscles in chronic Bright's disease is often a forerunner of general uræmic convulsions, and in acute rheumatism may suggest the onset of pericarditis. The working of the alæ nasi, so typical of pneumonia, is a sign, as Sir William Jenner used to put it, "of difficulty of entrance of air into the tissue of the lung."

In unilateral convulsions dependent on a lesion in the motor portion of the cerebral cortex, how important it is to observe the initial spasm, for this is our chief guide to the position of the lesion, and enables the surgeon to fix on a site for operation. Sometimes this "**signal**"

FIG. 19.—Face of Woman aged 64, the subject of Paralysis Agitans.

spasm begins in the face, as in a case reported by Berkeley, where persistent clonic spasms, chiefly of the zygomatic muscles, were found to be due to a small focus of superficial softening in the ascending frontal convolution opposite the inferior frontal sulcus.

In hysterical hemiplegia the face escapes paralysis, but is sometimes dragged to one side by a peculiar spasm of its muscles, which also affects the tongue, and curls its point round to the contracted side of the face. This deviation in position of the tongue and face towards the side on which the limbs are weak makes the case present a close

died rather suddenly after ejecting from the mouth a large quantity of blood, and the autopsy revealed not only pleuritic effusion, but an aneurysm of the descending thoracic aorta, which had ruptured shortly before death. Cover the faces of two persons suffering from disease in the abdomen; in both we find tenderness and resistance on pressing over the right rectus muscle. Uncover the faces, and we see that one patient has merely some functional or temporary affection, while the sunk, depressed features and earthy complexion of the other tell us that we have to deal with malignant disease in the neighbourhood of the pylorus, and that the prognosis solely consists in estimating the duration of life.

In serious illness, too, the face may give the first indications of approaching convalescence. Thus in a case of typhoid fever, the patient, previously delirious and semi-conscious, was found one morning with his eyes open, a brighter aspect, and able to talk intelligently; but it was not till two days later that the temperature began to fall and the pulse to become less frequent.

It is, however, necessary to remember that in many cases the face offers no positive indications; thus it is one of the commonest experiences in children's practice to find physical signs of pleuritic effusion when the face presents no marked departure from health; but such unchanged faces have also their value, suggesting, as they do, unimpaired vital forces.

ATTITUDE.

Attitude may be defined to be the posture or position of the body, or of any part of it, whether temporary or permanent, while in a state of inaction. The term is thus synonymous with "**fixed expression.**"

Attitudes the result of disease may be broadly separated into two classes, according as they are produced by normal or by abnormal muscular action.

Class I.—Attitudes Associated with Normal Muscular Action.—This class includes all the **instinctive attitudes**—that is, postures which are instinctively assumed by a patient in order to obtain relief from pain or other distress. It also includes cases in which the attitude is the direct result of some deformity of the skeleton; of these osteitis deformans is a notable example. In this group there is no necessary disease of the muscles or of the nervous system, although the elimination of disease in these situations may be difficult in some cases. Examples of attitudes belonging to this class may be considered under the following headings.

Decubitus, or Position of the Body in Bed.—While in health and in some diseases, a person in bed lies indifferently on the back or on

times a marked feature in pericarditis, the patient alternating between the back and the side position.

Station.—When a healthy person of normal conformation stands

FIG. 21.—Osteitis Deformans in a man aged 69. Duration of the disease, which came on after much exposure to cold and damp, about 35 years. The cranium, spine, and long bones of the extremities mainly affected. (Mr. Southam's Case, *Med. Chron.*, 1888.)

with his feet near together and his body erect, the line of gravity of the whole body falls in front of a line drawn between the two ankle-joints. But in a person who is ill, or afflicted with some deformity the axis of the body may deviate from the vertical, the centre of

arch and the iliac crest. This distance, which may be designated the height of the flank, is always less on the healthy than on the diseased side. A compensatory curve of the upper portion of the spine is frequently present, which may produce an elevation of the shoulder on the healthy side, this being again compensated for by a second curve (see Figs. 22 and 23). The primitive deviation of the spine in the dorso-lumbar region is always more marked towards the termination than at the commencement of a sciatica, and may persist, owing to permanent contraction of the muscles on the healthy side, long after the sciatica is cured. It is, therefore, important, in any case of lateral curvature of the spine, to admit the possibility that it may owe its existence to a present or past sciatica. In a few cases of sciatica, and especially when associated with a lumbo-sacral neuralgia, the spinal deviation is towards

FIG. 22.

FIG. 23.

Attitudes in Sciatica of the right side. (*Brissaud, Arch. de Neurol.*, 1890.)

the diseased side, the trunk muscles on this side being in a state of contraction. Then the height of the flank is greater on the healthy than on the opposite side. All the joints of the affected limb are flexed, and walking, and even standing, are difficult or impossible. If the patient is able to stand, he does so by throwing the weight of his body upon the healthy side. In order to accomplish this, he has to project the healthy hip outside a vertical line passing through the sole of the healthy foot, and the attitude is similar to that of a person carrying a heavy bucket of water, the diseased side corresponding to the side on which the bucket is carried (Figs. 24 and 25).

Class II.—Each of the attitudes referred to in the first class is the result of comparatively healthy muscular contraction, but we have now to consider deformities which are produced in consequence of an altera-

tion in the nutrition, tone, or strength of certain muscles. A study of the instinctive attitudes is one of much interest, and sometimes may be of help in diagnosis and prognosis, but its value is as nothing compared to that of the study of the present group, for in this group the attitudes are frequently the most important outward signs of the disease, and furnish us with reliable information as to the locality of the lesion.

It is difficult to form a satisfactory clinical classification—that is, to arrange muscular deformities into groups, each of which shall rest on a definite pathological basis. We might divide alterations of posture into those due to increased and those due to diminished action of the muscles of the part affected. In practice, however, we often find it very difficult to assign a case to its proper group—to decide, for

FIG. 24.—Attitude in Sciatica of the left side associated with Lumbo-sacral Neuralgia. (*After Brissaud.*)

FIG. 25.—Normal attitude when carrying a heavy bucket. (*After Brissaud.*)

example, whether a muscular deformity of the hand or foot is produced by excessive contraction of certain muscles, or by normal action of those muscles, their opponents being weak. When a limb is seized with convulsions, it is perfectly clear that any unusual position is due to violence of muscular action; but in chronic deformities such as those produced by rheumatoid arthritis, there is often a real difficulty in assigning a due proportion to the influence of excessive and diminished muscular contraction—that is, to muscular weakness and rigidity. The problem, too, may be mechanically complicated by the supervention of fibroid contractions about joints, and further by the actual shortening of even muscular structures.

Without attempting to give a complete explanation of, or even to enumerate all the vicious attitudes imposed upon the body or limbs

in consequence of abnormal muscular action, we shall arrange them in two groups, the first group comprising cases in which rigidity of muscular tissue is the most striking feature, the second group cases in which muscular weakness is most prominent.

Group I.—Abnormal Postures in which Muscular Rigidity is the Dominant Feature.—The most marked example of general rigidity is tetanus; in severe cases the characteristic attitude is that of **opisthotonos**, in which the vertebral column is arched backwards so that in the dorsal position the body rests on the head and heels. Opisthotonos also occurs in strychnine poisoning, in hydrophobia, and to a variable degree in cerebro-spinal meningitis. It is also a feature of the convulsions of hysteria, the excessive contraction of the extensor muscles sometimes bending the neck so far back that the vertex, or

FIG. 26.—Feet of young child the subject of Tetany—characteristic attitude.

even the forehead, becomes the anterior point of support. Rigid retraction of the head is also frequently present in cerebellar disease and in meningitis; boring of the occiput into the pillow being a prominent feature of chronic infantile meningitis affecting the posterior fossa of the base. It sometimes occurs as a result of rheumatism of the muscles at the back of the neck. **Pleurosthotonos** is a term applied to lateral flexion, **emprosthotonos** to arching forwards of the body—either may occur in tetanus and in hystero-epilepsy.

Tetany is a condition characterised by a peculiar tonic spasm of the extremities, which is generally symmetrical and lasts for a variable time. The wrists are slightly flexed, while the hand assumes the attitude of that of the accoucheur, the fingers being approximated, flexed at the metacarpo-phalangeal and extended at the other joints;

the thumb is extended and strongly adducted, its tip being applied to the radial side of the forefinger, and the palm is made still more hollow by an approximation of its inner and outer borders. The characteristic change in the foot is extreme flexion of the toes, which sometimes overlap one another; the forepart of the sole is often contracted so as to become remarkably concave with a median furrow, due

FIG. 27.—Attitude in Paralysis Agitans. The same case as Fig. 19.

to approximation of the outer and inner margins. The hands and feet are usually painful, tender and swollen, and the wrist and dorsum of the foot often red and shiny. The rigidity may spread to other parts of the limb, and even, in rare cases, to all the muscles of the body. In the eclamptic attacks occurring in infancy the hands and fingers are rigidly flexed at all their joints, and the attitude further differs from

that of tetany in the fact that the thumbs are mostly flexed and drawn inwards under cover of the firmly bent fingers. Infants affected with tetany are almost invariably rickety, are subject to attacks of laryngismus stridulus, and usually present great irritability of the facial muscles.

The rigidity of the muscles in **paralysis agitans** produces an attitude which is characteristic of the disease. The head and upper part of the body are strongly bent forward; the upper limbs are slightly flexed at all their joints, the wrists, however, being usually extended; the elbows are carried somewhat away from the trunk, while the hands are held in front of the abdomen. The posture of the hand closely resembles that of tetany, but the ring and little fingers are usually slightly flexed at the phalangeal joints, while the thumb, although extended, is not pressed firmly against the index finger, but is opposed to it, making the attitude similar to that adopted in holding a pen, or in rolling a cigarette. In the lower limbs muscular rigidity causes slight flexion

FIG. 28.—Position of Foot and Great Toe in Spastic Paraplegia.

of the hip and knee. At an advanced period of the disease the patient appears to move all of a piece, as if the joints were soldered together.

The various postures met with in cases of **spastic paralysis** are well worthy of study, for their recognition suggests that in all probability some portion of the upper segment of the motor tract has its functions impaired or abolished.

The rigidly flexed upper limb of hemiplegia is described in the chapter on the nervous system, but we would specially notice here the condition of the feet in chronic diseases of the spinal cord, when situated above the lumbar enlargement and involving the lateral columns. In such cases the heel is drawn up, owing to [illegible]id condition of the calf muscles, and the joints of the to[illegible] those of the great toe, are markedly hyper-extende[illegible] attitude is seen in the spastic bilateral hemiplegia[illegible] frequently the patient, when supported, stands [illegible] while the legs tend to cross one another, [illegible] traction of the adductor muscles of the th[illegible]

It is to be observed that the attitude of the hand in infantile hemiplegia differs from that in adult hemiplegia. In the latter the long flexor of the fingers is mainly affected by spasm, but in the former it is the interossei and lumbricales which are chiefly *rigid*, so that the fingers are flexed at the metacarpo-phalangeal joints and extended at the other joints. In the youngest infant this peculiarity may be brought out even when the contraction is slight in degree, by getting

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Group 2.—Abnormal Attitudes, in which Muscular Weakness is the Dominant Feature.—A familiar example of general muscular weakness is afforded by the dorsal posture seen in many acute diseases. Thus in the specific infectious fevers, when there is much prostration and mental apathy, the patient lies passively on his back with relaxed outstretched limbs and he tends to slip down towards the foot of the bed. This attitude, with loss of power to turn in bed, is especially noticeable in typhoid fever, in which disease it may occur as early as the second week; it conveys the impression of a loss of muscular tone, and the patient looks as if sinking through the bed.

This passive dorsal posture is also observed in typhus fever, in the collapse stage of cholera, in severe pneumonia, in coma and towards the fatal termination of many other diseases.

FIG. 30.—Position of Upper Limbs when all the muscles are paralysed, with the exception of those supplied by the 5th roots. (*Thorburn, "Surgery of the Spinal Cord."*)

Whenever there is weakness of the extensor muscles of the pelvis and spine, the forward inclination of the pelvis and lumbar vertebræ is compensated for by a posterior displacement of the upper part of the body, so that a plumb-line let fall from the most prominent of the spinous processes of the dorsal vertebræ clears the sacrum by an inch or more.

This hollow-back posture, called **lordosis**, is a prominent feature in pseudo-hypertrophic paralysis; it also occurs sometimes in infantile paralysis and in multiple peripheral neuritis.

Paralysis of the abdominal muscles also gives rise to lordosis; the abdomen protrudes and is flaccid, and there is a compensating backward curve of the upper part of the spine, but this does not project beyond the buttocks.

Examples of **deformities of the limbs** depending on wasting or weakness of certain muscles, their antagonists being comparatively or entirely spared :—

The various attitudes assumed by the upper limbs in lesions of the cervical enlargement of the spinal cord or its roots are of much interest and importance. Thus Mr. Thorburn reports a case of compression of the cord from a fracture-dislocation between the fifth and sixth cervical vertebræ—in which "all the muscles of the arms were paralysed, with the exception of the biceps, brachialis-anticus, supinator longus and deltoid ; the consequence being that the elbows were flexed, the shoulders abducted and rotated outwards, and the hands and arms fell into the position indicated in the annexed engraving, taken from a photograph."

Another example is **wrist-drop**, as a result of lead poisoning, or of pressure on the musculo-spiral nerve as it winds round the humerus.

FIG. 31.—From a Case of Lead Paralysis, in which the dropped hands were maintained in the supine position. (Dr. Ross's Case.)

In the latter case the supinator longus muscle is involved, as well as the extensor muscles on the back of the forearm, so that flexion at the elbow, when the hand is midway between supination and pronation, is feebly performed. This, which is sometimes called the drunkard's arm, is often caused by a person sleeping with his arm hanging over the back of a chair. In lead poisoning there is usually double, but there may be single wrist-drop, and the supinator longus muscle is unaffected. A patient with this deformity frequently enters the consulting-room with his hands not dropped, but held up in front of him and supinated ; such an attitude ought at once to suggest weakness of the extensor muscles of the wrists (see Fig. 31), but the unwary practitioner is prone to mistake it for spasm of the flexors.

Double wrist-drop, in conjunction often with **double ankle-drop**, is

hand certain of the thumb muscles appear to be specially affected ; the two phalanges are usually slightly flexed, while the metacarpal bone is

FIG. 35.—Showing normal action of thumb and little finger. (*Ross.*)

FIG. 36.—Showing action of thumb and little finger when the flexor brevis, abductor and adductor pollicis and opponens minimi digiti are feeble. (*Ross.*

drawn backwards so as to lie nearly in the same plane as the metacarpal bones of the fingers; frequently, too, it is approximated to the

FIG. 37.—First degree of Paralysis, from a case of Alcoholic Neuritis. (*Ross.*)

index finger. When the patient is asked to touch the tip of his little finger with the point of his thumb, it will be found that he is unable

FIG 38.—Second degree of Paralysis, from a case of Syphilitic Multiple Neuritis. (*Ross.*)

to do so except by bending the last phalanx of the thumb, and even then the side rather than the tip of the finger is reached (Fig. 36).

FIG. 39.—Third degree of Paralysis, from a case of Alcoholic Neuritis. (*Ross.*

A similar condition obtains in the foot. In the first degree of paralysis, the toes are hyper-extended at the metatarso-phalangeal joints

FIG. 40.—Fourth degree of Paralysis, advanced Alcoholic Neuritis. (*Ross.*)

and flexed at the phalangeal joints with the exception of the great toe, which is hyper-extended at all its joints, the attitude being almost

FIG. 41.—Showing adduction of first metacarpal as well as slight flexion of thumb at both joints, from a case of Chorea.

identical with that met with in spastic paralysis (compare Figs. 37 and 28).

In the second degree of paralysis the distal phalanx of the great toe

becomes flexed; and in the third degree its proximal phalanx also; while in the fourth degree of paralysis the other toes and the whole anterior part of the foot are loose, pendulous and curved down towards the sole.

The above deformities of the hands and feet sometimes occur during an attack of acute rheumatism or of chorea, but as a rule are less marked than in cases of alcoholic paralysis.

FIG. 42.—Claw-hand of girl, the result of Neuritis of left ulnar, and to slight degree of left median, following Scarlet Fever.

In chorea drawing backwards of the metacarpal bone of the thumb with flexion of the distal phalanges is common enough; sometimes the

FIG. 43.—Slight degree of Claw-hand in consequence of Rheumatic Neuritis. (Dr. Dreschfeld's Case.)

thumb is abducted, sometimes it is adducted, touching the radial side of the metacarpal bone of the forefinger (see Fig. 41).

The claw-hand (*main en griffe*) and the **claw-foot** (*griffe des orteils*)

result from weakness of the interossei and lumbricales. In these deformities the first phalanges are over-extended, but the distal phalanges are markedly flexed. The claw-hand is typically seen in certain cases of progressive muscular atrophy, and in spinal pachymeningitis, when the lowest part of the cervical enlargement is involved; in the latter case the wrist is hyper-extended. Injury or disease of the ulnar nerve may also produce this deformity, but not so completely, owing to the escape of the two outer lumbricales which are supplied by the median nerve (see Fig. 42). A slight degree of claw-hand is not uncommon in cases of sub-acute rheumatism (see Fig. 43). The claw-foot in conjunction with talipes equinus or equino-varus occurs in pseudo-hypertrophic paralysis and in Friedreich's disease.

Club-foot.—The several varieties of this deformity are met with in infantile paralysis. Talipes equinus and talipes equino-varus occur most frequently, talipes valgus also occurs, but talipes calcaneus is only

FIG. 44.—Infantile Paralysis, extreme Talipes Equinus.

rarely present. In one class of cases the deformity is associated with shortening and rigidity of some of the muscles, while in another class all the muscles are limp, and the paralysed limb dangles about like that of a doll.

Friedreich's disease is characterised by a special foot deformity. The plantar arch is greatly increased, and the toes have their first joints over-extended and their distal ones flexed; in standing, the foot rests on the heel and the prominent balls of the toes, and it is often possible to pass the hand beneath the sole. The heel too is drawn up to a varying extent; in short, there is a combination of talipes equinus, excavation of the sole and a claw-like character of the toes; the great toe is most affected, and indeed, a prominence of the extensor proprius pollicis has been regarded as an early sign of the disease, and therefore one of bad augury.

It may here be observed that the foot of ordinary **locomotor ataxy** presents a normal shape, and that when talipes equinus occurs in consequence of a complicating peripheral neuritis, there is plantar

flexion of the toes—that is, a position which is precisely the opposite of that observed in Friedreich's disease. It should be borne in mind too, when examining the feet of persons suffering from chronic diseases, that talipes equinus or equino-varus may be simply the result of pro-

FIG. 45.—Attitude of feet in Friedreich's disease.

longed confinement to bed, being not uncommonly noticed, for example, during the later periods of typhoid fever.

The Hand in Rheumatism.—Muscular atrophy is a striking feature in all chronic forms of rheumatoid arthritis; it is frequently present, and is sometimes prominent, in acute rheumatism (see Fig. 46). In

FIG. 46.—Extreme atrophy of hand muscles in a girl aged 8, probably from Rheumatic Neuritis. (Dr. Barlow's Case.)

these cases distortions of the hand and fingers are mainly the result of the action of comparatively healthy muscles whose antagonists are weakened. In chronic rheumatoid arthritis the common type of hand is one in which the wrist and metacarpo-phalangeal joints are flexed, while the first phalangeal joints are usually extended and the terminal one flexed. Many varieties of this deformity are met with,

owing to the varying degree to which flexion and extension of these joints takes place. Less commonly the first phalangeal joints are flexed and the distal ones extended, or both sets of joints may be extended.

When the terminal phalangeal joints are diseased the terminal phalanges are often twisted towards the radial side, and when the knuckles are affected the whole hand is deflected towards the ulnar side.

FIG. 47.—Chronic Rheumatic Arthritis, showing muscular atrophy and distortion.

WALKING.

When a healthy person is about to take a step in walking, say with the right leg, the weight of the body is first thrown on to the left leg, which becomes straight and rigid; this inclination of the body to the left elevates the right hip, while as the person gradually leans forward on the left foot, the heel of the right foot becomes raised, its toes resting for a moment on the ground till the gradually increasing flexion at the knee raises them from it. The foot is advanced forwards as the knee gradually straightens, and the toes would catch the ground if it were not for flexion of the ankle, which now takes place. The foot then, as it swings forwards, has its forepart at a slightly higher level than the heel, and it reaches the ground with the same inclination; hence the heel touches the ground first. In the meantime the left heel is being elevated, and then the left foot in its turn is swung forwards.

Of these movements the slight elevation of the forepart of the

advancing foot is perhaps the most important, for it is obvious that, if this movement is not properly performed, owing either to weakness of some muscles or rigidity of others, the toes must either rub along the ground or be cleared from it by an unusual degree of elevation of the hip or knee, or of both combined. The value of this movement is apparent when studying the many varieties in the normal gait produced by disease.

Abnormal Gaits, the Result Chiefly of Muscular Rigidity.

The Spastic Gait.—This is typically seen in spastic paralysis. The legs are rigidly extended, and in walking appear to drag behind the patient, the foot does not rise from the ground and makes a scraping or shuffling noise as it is hauled, not directly forwards, but forwards and outwards in a semicircular manner. The advance of each foot is indeed only rendered possible by an upward rotation of the pelvis, which throws the body first to one side, then to the other, and gives the gait a waddling character. The feet are often in-turned, and in aggravated cases readily get entangled, while in making an effort to free them the patient is elevated on tip-toes, and his lower limbs become the seat of violent tremor.

In hemiplegia, when there is much rigidity present, the paralysed leg is advanced in a similar manner, the toes during the forward semicircular movement making a characteristic scraping noise: but when there is much paralysis and slight rigidity, the leg is partly swung and partly dragged round by the inward rotators of the healthy limb.

In Paralysis Agitans, the body, as already mentioned, has a forward inclination; it appears as if solidified, and in walking moves as one piece. The steps are short, and at first often taken slowly and with difficulty, but the pace rapidly increases till the walk becomes a run, and the patient looks as if compelled to hurry forwards in order to recover his balance; some patients, if suddenly jerked backwards, begin to walk or run in this direction although their bodies are inclined forwards.

Abnormal Gaits, the Result Chiefly of Muscular Weakness.

The High Stepping Walk of Peripheral Neuritis.—This is well seen in many cases of alcoholic paralysis, and depends chiefly upon the flatness of the feet and the weakness of the anterior muscles of the leg. There is thus inability to flex the ankle, and when the heel is raised the toes drop, and are only prevented from trailing along the ground by an unusual degree of flexion of the knee and hip. The undue elevation of the knee of the advancing leg makes the gait resemble that of a high-stepping horse; and the drop of the toes at each step unduly

exposes the sole to the view of an observer standing behind the patient.

The Waddling Gait of Pseudo-Hypertrophic Paralysis is also largely due to inability to raise the fore part of the foot. But, in addition to this, it is to be observed that the balance in standing cannot be maintained unless the feet are widely separated, hence in walking the necessary elevation of the advancing foot is obtained, not so much by flexion of the knee as by an inclination of the body, first to one side, then to the other. This oscillation of the body, together with the shortness of the steps, makes the gait resemble the waddling of a duck.

The Hysterical Gait.—The necessity for a due amount of dorsal flexion of the advancing foot in normal walking has been already emphasised. In the normal gait the sole of the advancing foot presents a slight upward inclination from heel to toe; but in the spastic and paralytic gaits the upward inclination is in the reverse direction—so much so, indeed, sometimes, that the soles of the feet can be well seen by an observer standing behind the patient. Now, in hysterical paralysis, as a rule, there is no inclination of the sole; the heel is not elevated as in spastic paralysis, the fore part of the foot is not dropped as in peripheral neuritis, nor indeed is it so much raised as in health; the soles, in fact, are held close to and parallel to the ground as the patient shuffles cautiously forwards in a straight line.

Abnormal Gaits, Chiefly the Result of Muscular Incoordination.

Locomotor Ataxy.—In this disease it is often difficult for the patient to maintain the erect posture, and even at an early stage he may be unable to do so when his feet are close together and his eyes closed. At an advanced period of the disease he cannot stand without support, and it may be observed that all the extensor muscles of the body are in a state of powerful contraction; the resulting tendency to fall backwards is counteracted by a forward inclination of the body, which is supported by two sticks. In an uncomplicated case there is no muscular weakness, hence flexion of the ankle can be powerfully performed, and in walking this movement is usually more marked than in health. The gait is characterised by a quick and high elevation of the foot, which is suddenly and forcibly projected forwards and outwards, and then brought down to the ground with a stamp, the heel striking the ground first; hence these patients have been called "**stampers.**" In descriptions of the ataxic gait, too much stress has been laid on the heel coming down first, for, after all, this is a peculiarity of the normal gait; it is nevertheless true that the upward inclination of the sole of the projected foot from heel to toe is often

greater than in health, and in such cases stamping of the heels will be a marked feature, although perhaps stamping with the whole sole is just as common. In advanced cases walking becomes impossible and, even when the patient is well supported, the legs are thrown hither and thither in the greatest disorder.

The gait of **Friedreich's disease** is very similar to that of locomotor ataxy, but tremor is usually more manifest in the limbs, and progress, instead of being in a straight line, is frequently obtained in a zigzag fashion. In many cases the gait appears to combine the peculiarities of ataxy with those of the reeling gait of cerebellar disease.

CHAPTER IV.

TEMPERATURE.

THE temperature of the body is taken by means of the clinical thermometer, the bulb of which is placed, as a rule, in the axilla or mouth. In young children it is often more convenient to insert it within the fold of the groin, or to pass it into the rectum.

TEMPERATURE IN HEALTH.

In the axilla or mouth the average normal temperature is 98.6° F. In the rectum or vagina it is about half a degree higher.

At birth and during childhood it is slightly higher than at subsequent periods of life.

During the twenty-four hours variations of from 1°–2° F. (97.5° F.–99.5° F.) occur, the "daily maximum" usually being reached between five and eight in the evening, while the minimum record is made at midnight and during the early hours of morning.

TEMPERATURE IN DISEASE.

Any persistent elevation of temperature above 99.5° is indicative of some morbid condition. It must, however, be clearly recognised that very serious disease may be present with a temperature normal or even sub-normal.

According to the degree of pyrexia, febrile affections have been classified as follows :—

State.	Range of Temperature.
Hyperpyrexia	Above 105.5°.
High fever	Between 103–105°.
Moderate fever . . .	Between 101°–103°.
Slight fever	Between 99.5°–101°.
Normal temperature . .	98.6°, or between 97.5°–99.5°.
Sub-normal temperature .	Between 96°–97.5°.
Collapse temperature . .	Below 96°.

Abnormal temperatures show periodic daily fluctuations, which as a rule are similar to those observed in health; thus in the early morning there is usually a fall or **remission**, and in the evening a rise or **exacerbation**. Hence, valuable as a single record may frequently prove, accurate knowledge of the febrile process can only be ascertained by repeated observations. In hospital all temperatures are usually registered about 8 A.M. and again at 8 P.M., but in certain fevers it is desirable to have the temperature recorded on the charts every four hours, and in some cases even oftener.

Occasionally, as in some cases of tuberculosis, the fever type is inverted—that is, the temperature is lowest in the evening and highest in the early morning.

According to the differences between the maximum and minimum temperature occurring within the twenty-four hours, certain fairly distinct groups have been recognised :—

1. **Continued Fever.**—Here the temperature is above the normal, and the daily fluctuations are slight, and not greater than in the healthy adult.

2. **Remittent Fever.**—The daily variation is here greater than in the continuous pyrexia, but the minimum never reaches the normal level.

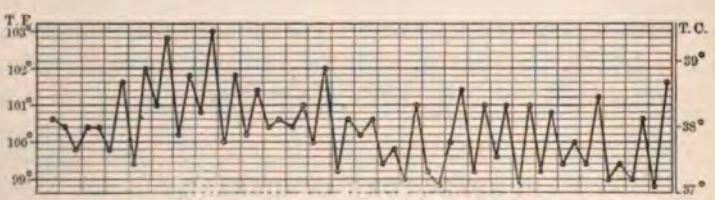


FIG. 48.—Leucocythæmia in a girl aged 8, showing remittent type of temperature.

3. **Intermittent Fever.**—In this form the minimum temperature falls to normal, or even below it, while the maximum is two or more degrees above it.

The Course of Pyrexia is divisible into three stages :—

1. The onset, period of invasion, or initial stage, during which the temperature commences and continues to rise.

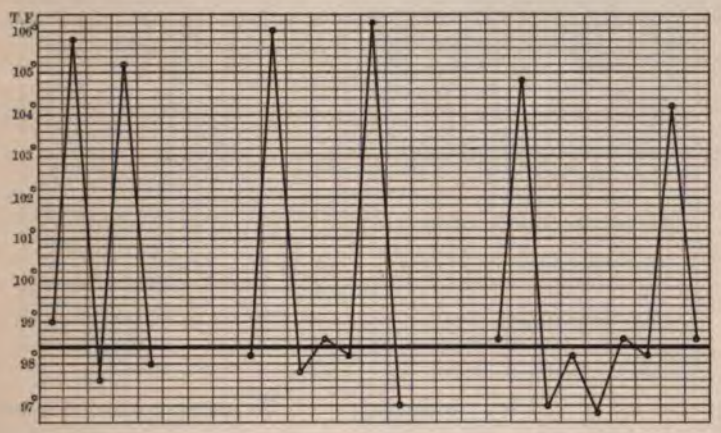


FIG. 49.—Intermittent type of Temperature in the different varieties of Ague.

2. The "fastigium," or period during which the temperature is maintained at its acme or highest level.

3. The issue, including the period of "defervescence" and recovery, or the stage of either hyperpyrexia or collapse, and approaching death.

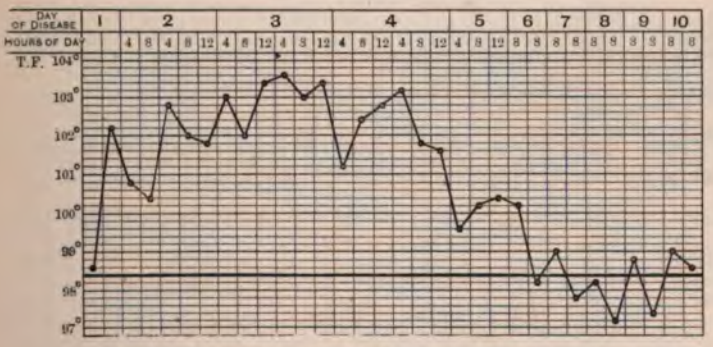


FIG. 50.—Moderate attack of Scarlet Fever in a boy aged 9.

The Initial or Pyrogenetic Stage.—The mode in which the temperature rises varies greatly in different diseases. Sometimes the rise is rapid and almost continuous, when it is often associated with shivering or *rigor*. In ague the maximum is often attained in a few hours, and in scarlet fever (see Fig. 50), erysipelas and croupous pneumonia

(see Fig. 51) within a day or two. Occasionally the ascent is gradual and the acme is reached by a series of oscillations extending over three

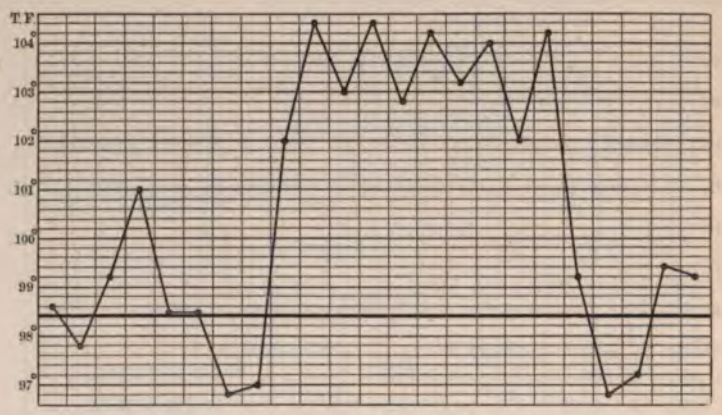
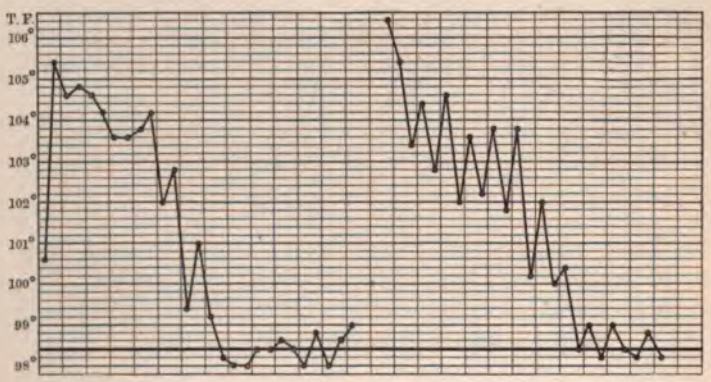


FIG. 51.—Boy aged 2½, admitted to Hospital with Dropsy and Albuminuria following Scarlet Fever. On the fifth day he began to cough, and at night the thermometer registered 104.4°. Two days later physical signs of *Pneumonia* were detected at left base. The crisis occurred on the sixth day of the attack. (On this chart each division represents half a day.)

or more days; this usually happens in typhoid fever and in tuberculosis, and in a slower and more irregular manner in acute rheumatism, pleurisy and pericarditis. The ascent in typhoid is usually stated to be characterised by a rise each evening of two degrees with a morning

FIG. 52.



Pneumonia of right upper lobe in a boy aged 6. Pneumonia of left upper lobe in a girl aged 11.
In both cases defervescence by *lysis*, the temperature becoming normal about the eighth day.

fall of one degree, a temperature of 103° to 104° F. being reached about the fifth day; but such a course is by no means constant, and

occasionally the thermometer in the evening even of the first day may register as much as 103° F.

The Fastigium, or course of the fully established pyrexia, must be

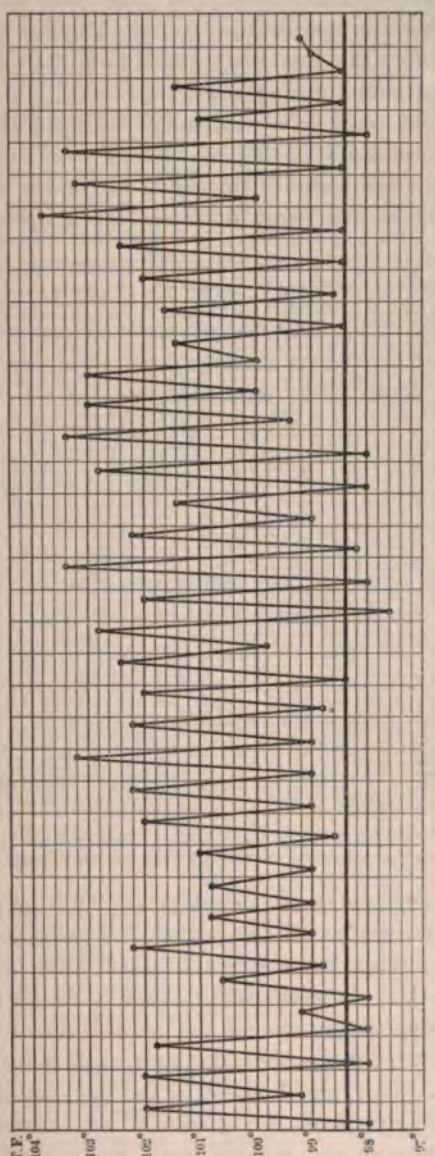


FIG. 53.—Portion of Temperature Chart of a boy aged 5, the subject of Tubercular Peritonitis. The autopsy revealed an extreme degree of Tubercular Peritonitis, a few tubercles in the intestine, and much tubercular disease in the lungs: no tubercles elsewhere.

studied in respect to its height, its character, the extent of its daily variations and its duration.

A **high** temperature is not always in itself a serious omen. Thus in typhus a high continuous temperature of from 104° to 106° may be regarded as normal to the disease. But in typhoid it is almost always a grave feature, and speaking broadly, in most fevers and acute inflammations the higher the temperature and the slighter the daily remission, the more serious the case. In typhoid fever, for example, if there are daily great variations during the second week, it is probable that the disease may terminate before the end of the third week; but if the daily variations are slight during the second week, then we cannot expect the fever to terminate till the end of the fourth week.

Pyrexia may be of but short **duration**, the temperature reaching

FIG. 54.—Pneumonia of right lower lobe in a girl aged 6. Defervescence by crisis on the sixth day; a "*pseudo-crisis*" occurring during the fourth night.

its maximum on only one or two occasions and then falling to normal. This is observed in ague, in chicken-pox and in some cases of erysipelas. More commonly a high temperature is maintained for many days, or even several weeks. In most acute illnesses, as scarlet fever, measles, erysipelas and croupous pneumonia, the pyrexia ceases within a week or ten days. In typhus the fastigium lasts until towards the end of the second week. In typhoid a fall in the temperature curve may sometimes be observed about the middle of the second week, but a normal point is usually not reached until the end of the third or fourth week. In some affections pyrexia may persist for weeks, or even months. Thus chronic pyrexia is met with in tuberculosis (see

Fig. 53), syphilis, malaria, rheumatism, leucocythemia, lymphadenoma, pernicious anæmia and suppurative processes.

The issue may be (1) favourable or (2) unfavourable. In a **favourable** case the decline or defervescence of the fever takes place either by crisis or lysis. In *crisis* there is a rapid fall of temperature, often as much as four or five degrees, to the normal, or even below it, in from twelve to thirty hours. This mode of termination is the rule in croupous pneumonia (see Fig. 54), ague, relapsing fever and chicken-pox. Occasionally an exacerbation of temperature occurs immediately before the crisis; it is spoken of as a "*critical perturbation*." In *lysis* the fall of temperature is gradual, taking several days to reach a healthy standard. Lysis occurs in typhoid, in broncho-pneumonia, in acute articular rheumatism and in scarlatina.

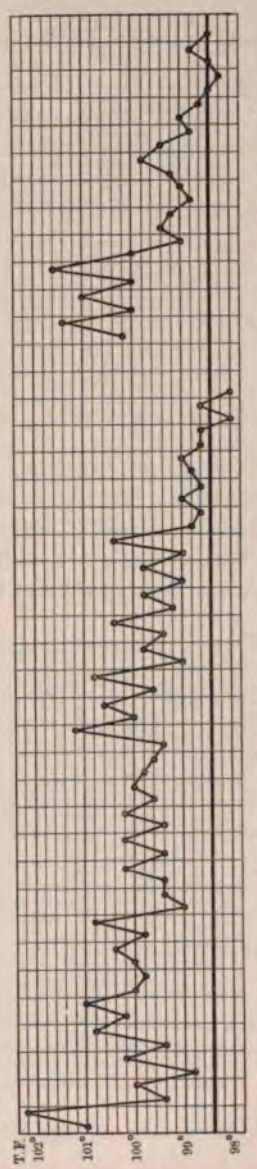


FIG. 55.

Between crisis and lysis there is, however, no abrupt line, and in many acute diseases the decline of the pyrexia follows an intermediate method. For example, in typhus there is often a gradual fall of temperature—usually about three days in duration. Occasionally there is a temporary fall towards or even to normal; this is spoken of as a "*pseudo-crisis*" (see Fig. 54).

(2.) The **unfavourable** issue follows either an *ascending* type, leading to the condition of "hyperpyrexia," or a *descending* type, in which a falling temperature is associated with symptoms of collapse.

Any sudden or considerable rise of temperature, while possibly due to a slight cause, is frequently indicative of the onset of some important complication; sometimes it is the forerunner of a fatal termination. The elevation may occur during the fastigium, defervescence or early

convalescence. It must, however, be remembered that important complications may arise without any perceptible elevation of the temperature; this is particularly the case in acute rheumatism, where the onset of pericarditis or the implication of a fresh joint is frequently unattended by any increase in the temperature. In not

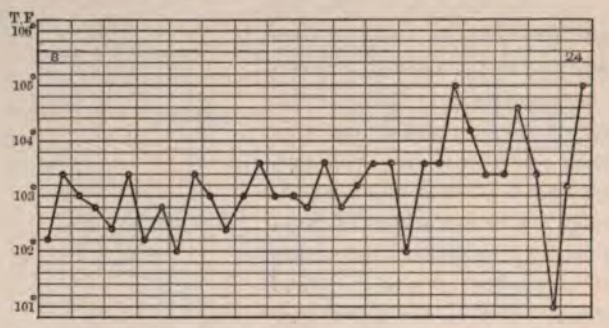


FIG. 56.—Typhoid Fever in a boy aged 6; death occurred on the twenty-fourth day.

a few instances, however, even in the absence of any recognisable complication, the temperature runs riot, and ascends, often with considerable rapidity, into hyperpyrexial ranges. In acute rheumatism hyperpyrexia is a well-known and often fatal complication. It occurs also in typhoid and in connection with certain cerebral lesions,

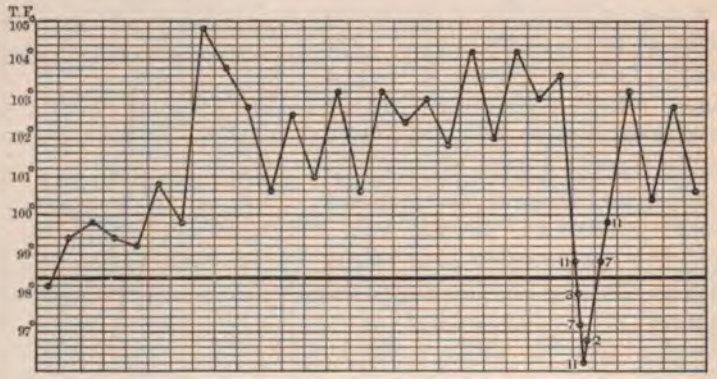


FIG. 57.—Relapse of Typhoid Fever in a girl aged 6. The temperature fell suddenly during one night, and was 96.2° at 11 A.M. The child complained of severe *abdominal pain* two hours later. Death two days later. Extensive peritonitis from intestinal perforation.

particularly hæmorrhage into the pons or medulla. It also follows on severe burns, or as a consequence of heat or sun-stroke. Extremely high temperatures have been recorded in neurotic cases, but as in many of these instances associated symptoms of excessive pyrexia were

not marked, there is the possibility that such "paradoxical temperatures" are factitious.

Far more frequently an unfavourable issue takes the descending type. Here the falling temperature, which in many instances occurs more or less suddenly, is associated with the characteristic symptoms of collapse. When met with in typhoid, it usually indicates intestinal hæmorrhage or perforation. Collapse temperatures occur also in pneumonia, usually in direct association with cardiac failure, and sometimes in acute tuberculosis.

Occasionally the temperature, taken at hourly intervals, gives no warning of a fatal termination, death occurring quite suddenly. (See Fig. 58.)

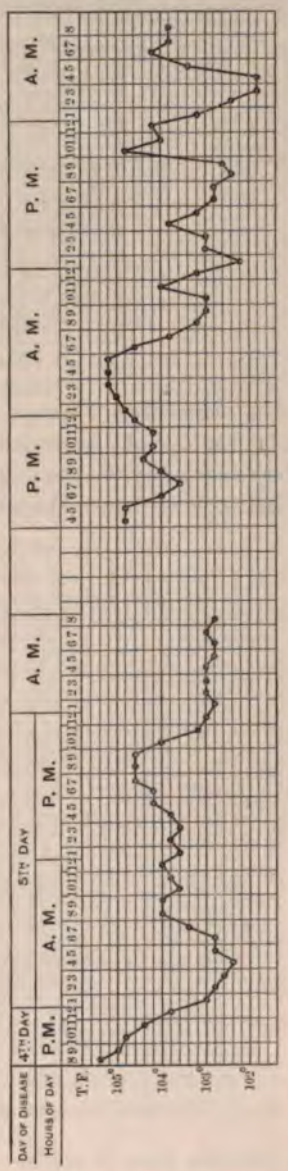


FIG. 58. Apical Pneumonia in a boy aged 5, terminating fatally on the sixth day. Apical Pneumonia in a girl aged 7, with fatal termination.

Diagnostic Value of Pyrexia.—It is seldom possible to diagnose with certainty the exact nature of any disease from observation of the temperature alone. It is true that in many affections, such as ague, pyæmia, typhus, typhoid, relapsing fever, scarlet fever and croupous pneumonia, the temperature frequently runs a more or less characteristic course; and that a study of a large number of cases of almost any febrile disease brings out certain salient features in the temperature curve which, though not pathognomonic in themselves, are, nevertheless, of great help, in conjunction with other symptoms, in forming a diagnosis of an obscure case of illness. But modifications of such salient features are very numerous, and it must be borne in mind that a pyrexial temperature is much more unstable than a normal one; it may, for example, be modified by a change in the patient's diet. A

slight pyrexial condition, occurring usually in the evening, is of considerable diagnostic importance in doubtful cases of tuberculosis. In many phthisical subjects the thermometer will render much assistance in forming a correct opinion as to the activity of the process.

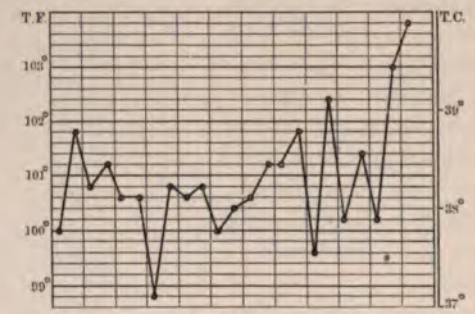


FIG. 59.—Tubercular Meningitis in a boy aged 3.

The temperature curve of a febrile disease is often much influenced by (1) the age of the patient; (2) by previous states of health or by idiosyncrasy; (3) by the presence of complications.

Influence of Age.—The temperature is much more readily disturbed

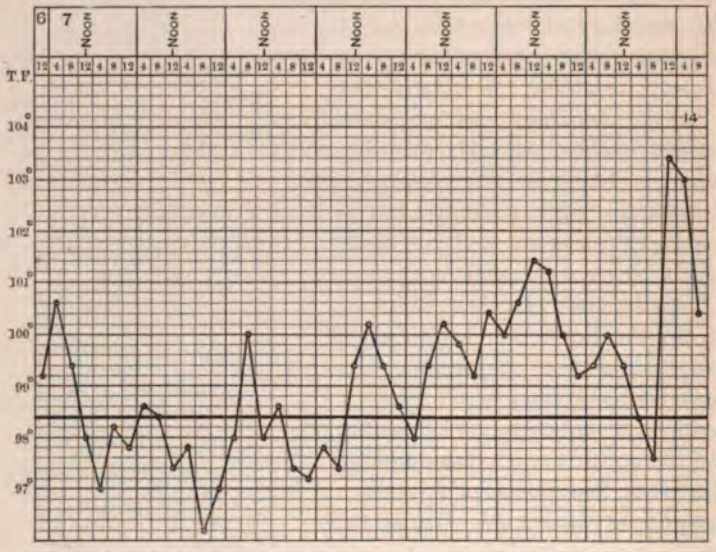


FIG. 60.—Tubercular Meningitis in a girl aged 14 months. Death on the fourteenth day.

in children than in adults; it is less subject to the ordinary laws of pyrexia and hence tends to run a more erratic course. Intermittent pyrexia of irregular type, lasting a few days and attaining each evening

a maximum of 103° or 105°, is particularly characteristic of slight intestinal catarrh or other minor ailments of early childhood. In serious illnesses, too, there are commonly an earlier rise and a somewhat higher temperature in the child than in the adult; frequently also the duration is shorter. Thus in children the fever of typhoid is often characterised by a sudden rise, a short fastigium and a quick defervescence. In advanced life and also in infancy, an opposite condition appears to obtain. A serious illness, such as pneumonia, may be attended by only a slight (see Fig. 61) or moderate degree of pyrexia, and hence it is especially necessary to thoroughly investigate any case of febrile illness at these two periods of life.

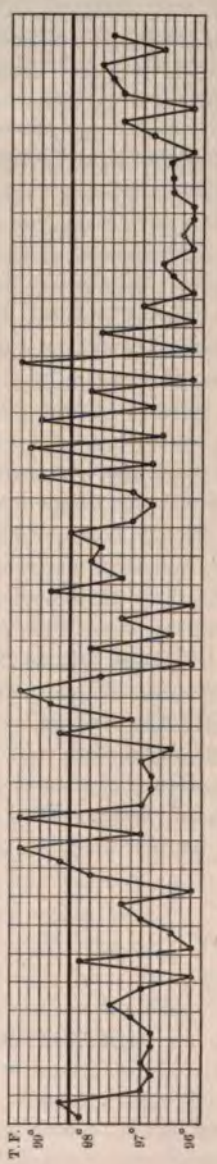


FIG. 61.—Pneumonia of right lower lobe in a marasmic infant aged 13. Complete recovery.

Previous State of Health and Idiosyncrasy.—The apparent peculiarity of the two extremes of life with respect to temperature just alluded to may be more dependent on lowered vitality than on actual age; at any rate, it often happens that vigorous infants and vigorous old people have attacks of pyrexia as severely as those of an intermediate age. Adults also present great differences with regard to the degree of pyrexia accompanying maladies of apparently identical severity. Some of the differences may be explained by the previous state of health of the patients. Thus the pyrexia of pneumonia in an alcoholic subject often runs a protracted course with a daily elevation of temperature only moderate in degree; whereas in a previously robust subject the temperature is usually higher, but its duration shorter.

In many cases, however, idiosyncrasy appears to be the only available explanation of certain exceptional pyrexial conditions.

Influence of Complications.—As has been indicated above, a sudden or considerable rise of temperature during the fevered state often indicates the onset of some complication. An abrupt and considerable fall, if not due to a natural termination or favourable issue, is also significant of some complication. More

moderate variations of the temperature should also be closely watched. Thus a hectic type of temperature may be observed after the crisis of pneumonia, owing to the development of an empyema. Elevation and persistence of temperature after incomplete subsidence of a fever may occur as a *recrudescence*. This is common in typhoid fever, in consequence of implication of fresh glands or of advancing intestinal ulceration. Elevation and persistence of temperature after a period of complete apyrexia is spoken of as a *relapse*.

Subnormal Temperatures.—Although there is no distinct proof that a condition exists which corresponds to the reverse of what we know as pyrexia, yet a subnormal temperature occurs in a number of well recognised morbid conditions. It is met with in chronic forms of heart disease, in diabetes, in marasmus, in myxœdema, and in many varieties of mental disease. Sometimes chronic phthisis is attended with a normal or subnormal temperature, but in this, as in other chronic diseases, brief periods of slight pyrexia are readily overlooked. In chronic forms of phthisis particularly, the temperature should be taken many times during the twenty-four hours before it can be affirmed that fever is absent. The presence of slight intercurrent elevations of temperature implies either that a fresh deposit of tubercle is taking place, or that some portion of the lung is the seat of an inflammatory process; but if the temperature remains normal or subnormal at all periods of the day, then it is highly probable that the progress of the disease is arrested or insignificant.

Collapse Temperatures.—Extraordinarily low temperatures have been recorded in sclerema neonatorum, the thermometer in the rectum registering 85°, 83°, or even lower. Collapse temperatures occurring suddenly during a febrile illness, if not due to recovery by crisis, are significant of a serious complication, as perforation of the intestine in typhoid fever. Very low temperatures have been registered in cases of fracture dislocation of the spine with injury to the cord.

CHAPTER V.

EXAMINATION OF THE SKIN AND ITS APPENDAGES.

The points to be observed in an examination of the skin may be considered under the following headings:—Colour; dryness or moisture; the presence or absence of eruptions or other lesions and their characters, if present.

CHANGES IN THE COLOUR OF THE SKIN.

Deviations from the natural rosy, somewhat diffused tint of the healthy face are innumerable, and often baffle description; yet changes in kind, depth and distribution of colour, in association with other conditions of the features, furnish to the keen observer a picture of the greatest diagnostic and prognostic importance.

Pallor of Skin is not always a sign of anæmia, while red weather-stained cheeks may distract our attention from bloodless lips and palpebral conjunctivæ. In the pallid face of large white kidney the lips usually show some degree of redness, and even if distinct œdema of the face be absent, the disease may be distinguished from simple anæmia by the watery, glistening appearance of the conjunctiva. Further, the colour of the simple anæmia of young women is generally different from that of pernicious anæmia. The whiteness of the former is commonly more marked, and if not a pure white, the blended tint is often greenish, while in pernicious anæmia a yellowish tinge is characteristic, and at the first glance appears to affect also the conjunctiva; but a careful examination shows that this in many cases is due to the shining through of the subconjunctival fat. The face of pernicious anæmia closely resembles in colour that of malignant disease of the abdomen, and particularly of cancer at the cardiac end of the stomach; but in the latter case the eyes are much sunk, the orbital ridges stand out, and there are lines of pain and depression around the mouth.

The pallor of aortic regurgitation, especially noticeable on the forehead, is distinguished from other pallors by its association with extensive visible arterial pulsation; this is also seen in the neck in anæmia, and when there is much arterial degeneration, but in aortic regurgitation not only is the whole length of the external carotid seen to pulsate, but also the temporal and often the facial artery. Extreme pallor sometimes comes on in cases of old valvular heart disease, in consequence of the supervention of large ulcerating vegetations.

Increased Redness of Skin is common in mitral stenosis. The colour in girls is often a bright red; in adults the face may be pallid, and have a tired and worn expression, in these respects resembling the face of phthisis; but small varicose venules, absent in the latter, are usually present in mitral stenosis. When there is regurgitation through the mitral orifice, the red is mixed with blue, and other causes of dilatation of the right side of the heart, such as emphysema or capillary bronchitis, are also characterised by purplish cheeks. The *bluish element* reaches its maximum in congenital pulmonary stenosis, where the conjunctivæ are of a light claret colour, the lips blackish-blue, and

the nose and cheeks of a deep purplish hue. In acute pneumonia a dusky red flush is seen over the malar bones, and is often more marked on the diseased than on the healthy side. In cerebral meningitis the forehead and ears may present a dusky red colour.

Yellow Skin.—A yellow discoloration of the skin is frequently seen in new-born infants; it is of about a week's duration, and is unattended by any signs of ill-health. A yellowish tinge is seen in pernicious anæmia, a greenish-yellow tint in chlorosis, a greyish-yellow hue in the cancerous cachexia, while in granular kidney disease, especially if associated with lead poisoning, the skin has a dusky yellow appearance.

In the above cases the conjunctiva is clear, and the colour of the urine is not materially altered; but in true **jaundice** or **icterus**, the conjunctiva and other mucous membranes are coloured yellow, and the urine presents various shades of yellow or green. In slight cases of jaundice the sclerotic conjunctiva is alone affected, and in severe jaundice, when the whole of the skin is markedly and pretty uniformly coloured, the yellowness is always especially marked in the conjunctiva. The inside of the mouth, and to a less degree the lips and tongue, also present a yellow colour. According to the intensity of the jaundice, the colour of the skin is a pale yellow, a deep orange, or an olive-green; in extreme cases it may become a brownish-green. The colour is usually deeper in old, emaciated, or dark-complexioned persons than in the young and fair. A persistent dark greenish-yellow skin is significant of permanent obstruction of the common bile duct, which is usually, but not necessarily, of cancerous origin. In commencing jaundice the urine and conjunctiva may be affected simultaneously, and sometimes the urine is deeply stained for a short time before the conjunctiva; while in temporary cases the colour may remain in the skin for some time after it has disappeared from the urine.

In association with jaundice, the patient often complains of a bitter taste and an itching skin.

THE MOISTURE OF THE SKIN.

The amount of sensible perspiration is very variable in health, and in different individuals exposed to the same conditions. An excess of perspiration, called **hyperidrosis**, may be general or local. General sweating accompanies a fall of temperature in febrile diseases, or is the result of general weakness and exhaustion. Thus profuse sweats occur towards the end of typhoid fever and during the early convalescence of scarlatina: they attend the decline of pneumonia, occur after the rigors of pyæmia and follow the hot stage of ague. When

sweating occurs at the commencement of febrile diseases, it usually indicates great weakness; but in acute rheumatism profuse sweats are characteristic of the disease, and are met with both early and late. In children, however, suffering from rheumatic fever, sweating is not common, and may be represented only by a slight increase in the moisture of the palms and soles. Profuse nocturnal perspiration is a feature of phthisis, in the early as well as in the advanced stages. Head sweating is an important sign of early rickets, and probably bears some relation to the hyperplastic skull changes. Sudden outbreaks of sweating have been noticed in epileptics; copious sweating occurs, too, during the course of an epileptic fit, but to a less degree than in tetanus, where, owing to the violent muscular action, perspiration streams from the body. The respiratory distress of asthma and the agonising pain of angina pectoris are also accompanied by hyperidrosis. In paralysis agitans the least exertion of mind or body is apt to produce intense perspiration.

Local hyperidrosis may be limited to the palms and soles, or be hemiplegic or paraplegic in distribution. It is probably dependent on faulty innervation, produced by a local neuritis, or by damage to the sympathetic system, as when the cervical portion of the spinal cord is injured, or by a brain lesion. Occasionally unilateral sweating occurs in hysteria.

A diminution or absence of perspiration, called **anidrosis**, may be observed in the early stages of many fevers, in diabetes and in granular kidney. In rheumatic fever, sudden drying of a previously moist skin is sometimes the forerunner of hyperpyrexia. A stinging, hot, dry skin is particularly noticeable in acute pneumonia.

Qualitative Changes.—Sometimes, in cases of jaundice, the sweat has a yellowish tinge. In uræmia and in cholera the quantity of urea may be so great that white crystals like hoar-frost are deposited on the body. Red or bloody sweat is occasionally met with, and in rare cases the sweat is tinted with blue or some other colour.

The secretion may be very offensive, as is frequently the case in the sweating feet of domestic servants. Rheumatic fever, syphilis and uræmia often give a characteristic odour to the sweat.

The **reaction** in health, when proper precautions are taken, is alkaline or neutral. In acute rheumatism the reaction to litmus paper may vary in different parts of the body; the administration of salicine preparations probably tends to convert an acid into an alkaline secretion.

CUTANEOUS ERUPTIONS.

By ROBERT B. WILD, M.D. (Lond.).

In considering the pathological changes in the skin, which give rise to the variations in its appearance or texture known as "skin diseases," it is necessary to remember that the skin is a complex organ, which may be roughly described as a fibrous tissue base, with a papillated surface, covered by numerous layers of epidermic cells; the deepest of these are columnar in shape, the superficial flattened into scales. Each papilla is occupied by a vascular loop or the termination of a sensory nerve, and in the fibrous tissue are embedded the appendages of the skin, viz., hair follicles with hairs and sebaceous glands, and the sweat glands; the shafts of the hairs and the ducts of the glands penetrate the epidermis to reach the surface of the body.

For the examination of the skin certain methods must be adopted.

(1.) The patient must be placed in a good light.

(2.) As much of the skin as possible should be seen at once.

(3.) The consistence of the implicated skin, and the effect of pressure upon the colour of a spot, should always be ascertained.

(4.) A pair of forceps should be used to extract hairs and remove scales or crusts, in order to expose the diseased surface to examination.

(5.) A microscope with a few reagents, such as liquor potassæ and ether, should be available for the examination of hairs, epidermic scales, or scrapings of the skin.

Elementary Lesions.—All forms of skin diseases are manifested by certain changes in the appearance of the skin. These changes may be of infinite variety, but on careful examination they may all be shown to consist of a small number of "elementary lesions" variously combined and grouped, just as an infinite variety of words can be made up by the various combinations of a small number of letters. These "elementary lesions" form, as it were, the alphabet of Dermatology, and it is necessary for the student to become thoroughly acquainted with their characteristic appearances and names.

The elementary lesions are divided into "**primary**" and "**secondary**," the former are caused directly by morbid processes in the skin, the latter owe their peculiar characters to further changes in the diseased patches after their formation.

Primary Lesions.—**Spots** (**maculæ**) are alterations in the normal colour of the skin over a limited area, extremely variable in size, shape and colour, and not raised above the general surface. When due to hæmorrhage, they are called *petechiæ* if small, *ecchymoses* if large, and *vibices* if in the shape of streaks.

Pimples (**papulæ**) are solid elevations of the skin, 5 mm. or less in diameter, and of variable shape and colour.

Nodules and **Tumours** are not very sharply differentiated; both are solid elevations of the skin larger than a papule. The term nodule is generally used for a cellular infiltration, such as tubercular or syphilitic nodules, while the term tumour is applied to all new growths, such as cancer or fibroma.

Vesicles (**vesiculæ**) are elevations of the skin, containing serous fluid, and less than 5 mm. in diameter.

Blebs (**bullæ**) are vesicles over 5 mm. in diameter.

Pustules are elevations of the skin containing pus.

Wheals (**pomphi, urticæ**) are solid elevations of the skin, only slightly raised above the general surface, but of comparatively large area; the size and shape are variable, and the colour white or pink.

Secondary Lesions.—**Scales** (**squamæ**) are thin plates of variable size, consisting of dead epidermis cast off from the surface of the skin.

Crusts (**scabs**) are solid masses formed on the surface of the skin by the drying of various fluids, such as serous exudation, pus, blood, or the secretions of cutaneous glands.

Fissures (**rhagades**) are linear cracks in the skin, involving the epidermis alone, or extending to the true skin.

Excoriations are localised lesions, of varying extent and depth, due to the mechanical removal of the epidermis by scratching or similar violence.

Ulcers are localised lesions of varying extent and depth, involving loss of substance of the true skin, and produced by disease.

Scars (**cicatrices**) are new formations of connective tissue which replace portions of skin destroyed by previous disease or injury.

To determine the nature of any case of disease of the skin, the individual lesions must be carefully examined as to whether the eruption is uniform, *i.e.*, presenting only one form of lesion, or multiform, *i.e.*, presenting two or more forms of lesion. In the latter case the lesions must be compared, and the secondary, if present, distinguished from the primary, so as to obtain a history of the course of the disease from its first appearance to its most advanced stage.

The nature of the changes in the skin being thoroughly ascertained, the first step in the diagnosis is to determine whether the disease is a local one of the skin, or whether the cutaneous eruption is only a local manifestation of a general disease. The distinction between these two classes is most important, inasmuch as the latter class includes many of the specific fevers, an early recognition of which is of the utmost importance, not only for the proper treatment of the patient, but also for the protection of the community. Another broad distinction

between the two classes is found in the fact that the treatment of the latter is general in character and without reference to the condition of the skin, while in the former class local treatment is of primary importance, and internal or general treatment adjunct to it.

The points to be ascertained in order to determine to which of the two classes a given case belongs are—(1.) The nature of the lesion or lesions. (2.) The distribution of the eruption. (3.) The length of time since its first appearance, and the manner in which it appeared—for example, whether a number of spots appeared simultaneously, successively, or in crops. (4.) The condition of the mucous membranes of the eye, nose, tongue and pharynx. (5.) The temperature of the axilla, mouth, or rectum. (6.) The condition of the internal organs.

From these data an accurate judgment can in most cases be formed, but not in all, as the time may have been too short for the characteristic features of a specific disease to manifest themselves; it is therefore needful to suspend a decision in doubtful cases, and to keep the patient under close observation until the course of development throws light upon the real nature of the disease.

If the symptoms of the patient point to the presence of a general disease, the student should then consider its nature. We therefore give a short account of those general diseases attended by cutaneous manifestations, with especial reference to their diagnosis; and for this purpose we classify them according to the appearances presented by the skin. By comparing the case before him with each of the general diseases in turn, the student will readily form a correct diagnosis.

If, however, the appearances and symptoms exclude any form of general disease, the case is a local disease of the skin, and the next step in the diagnosis is to determine whether the lesions are due to cutaneous parasites. For this purpose the following points must be observed:—(1.) The presence of parasites on the skin or hairs, or in the scales or scrapings from the surface. In most cases microscopic examination will be decisive; but some parasites can only be demonstrated by special methods of staining, and a diagnosis has to be made from other grounds. (2.) The character of the lesions. (3.) The distribution of the lesions. (4.) The history of the case. (5.) The presence of characteristic lesions in other parts of the body.

If the presence of some parasitic disease is probable, the diagnosis is completed by comparing the case under examination with the various parasitic diseases, until its exact nature is determined. If the result of careful examination is to exclude parasitic diseases, the diagnosis of the remaining local diseases of the skin is much simplified; the lesions are carefully examined, and the most advanced stage present is then taken

as a guide to diagnosis, and by comparison with the following account of the skin diseases, the nature of a given case can be readily determined. It is necessary for the student to remember that the classification adopted has no pretensions to a scientific arrangement of the diseases of the skin; it is merely a convenient arrangement for the purpose of facilitating diagnosis at the bedside or in the out-patient room, and it includes only those forms of disease which the student is likely to meet with in this country.

Finally, the student must be aware of the fact that certain drugs circulating in the body produce in some people eruptions on the skin which cause difficulty in diagnosis if the cause be not suspected and the previous history of the patient unknown. It is well, therefore, for the student to inquire whether the patient has been taking medicine, and if so, for what disease, and to make himself familiar with the appended list (p. 125) of the commoner drug eruptions.

Class I.—General Diseases with Cutaneous Lesions.

(a.) The lesions in the skin are local changes in colour, not elevated above the surface.

1. **Addison's Disease** ("Bronze Skin").—The tint varies from yellowish-brown to nearly black; the parts chiefly affected are those parts of the face and neck exposed to the sun, the backs of the hands, the axillary, umbilical and genital regions, and inner side of the thighs. The patches vary in size, and the colour usually shades off into that of the surrounding skin. Similar pigmentation is often found on the mucous membrane of the mouth. The other symptoms are marked anæmia, with increasing weakness and prostration.

2. **Chloasma Uterinum** is a discoloration of the skin in women symptomatic of pregnancy or of uterine disease; its typical form consists in a brown band across the forehead and spreading over the temples, fading at the margins into normal skin. Similar patches are found along the linea alba, round the nipples, and occasionally on the cheeks and other parts of the body.

3. **Graves's Disease** often presents patches of brown pigment in various parts of the body, especially those parts which are normally darker in colour; white patches are occasionally found associated with more deeply pigmented skin. The other symptoms are the prominent eyeballs, enlarged thyroid gland and cardiac palpitation.

4. **Scarlet Fever.**—The rash appears on the second day of the disease, usually on the upper part of the chest and neck, spreads to the abdomen and back, and finally to the limbs; the colour is bright red, but may vary in tint. Examined closely, the rash consists of

minute red spots, bright in the centre; if numerous, the coalescence of the spots gives a uniform red colour to the skin; if less numerous, a punctiform appearance. The face is flushed, but does not show individual spots. High temperature, sore throat, swollen cervical glands, are present, with more or less constitutional disturbance. After the first week the rash has faded away, and the skin presents a coarse or branny desquamation, which may be slight or very copious, and continue six weeks or more.

5. **Septicæmia, Relapsing Fever, Epidemic Cerebro-Spinal Meningitis, Cholera, Dengue.**—These diseases have no typical rash, but in many cases various eruptions appear as red spots or petechiæ. These eruptions are not essential characters, their distribution is irregular, and the diagnosis rests upon the general symptoms alone; it is, however, necessary to recognise their occurrence in order to prevent mistakes in diagnosis.

6. Macular eruptions also occur as part of the cutaneous lesions in **typhus fever and small-pox**; the other lesions are, however, of much greater importance, and will be described later on.

(b.) The lesions in the skin are raised above the general surface. Vesicles or pustules are not formed.

Rötheln, Epidemic Roseola, German Measles.—The rash appears on the first or second day in the form of pink spots, very slightly raised and varying in size, so that when small they resemble scarlet fever, and when large measles. The face is first affected, then the trunk and limbs. After about two days the spots gradually fade and leave a slight stain. Desquamation, if present, is slight. Catarrh of the mucous membranes and sore throat may both be present. Fever and general symptoms are slight or absent.

Morbilli (Measles).—The rash appears on the fourth day, first on the face, which is often puffy and slightly swollen, as small red spots, which rapidly become raised papules, 2–6 mm. in diameter, and of a darker red colour than the eruption of scarlet fever. The papules fade on pressure, and have a tendency to form groups of various size, which have a more or less crescentic shape. The trunk and limbs are affected to a variable extent. The rash persists for two or three days, then fades, leaving a slight staining of the skin for a few days, and often a little desquamation.

In a few cases (malignant or black measles) hæmorrhage occurs into the papules, which become purple or black and no longer fade on pressure.

The onset is usually acute, and the constitutional symptoms consist of pyrexia, with catarrh of the eyes, nose and respiratory tract.

Typhus Fever presents two forms of rash, which are both present at

the same time. (*a.*) The subcuticular mottling, seen best on the flanks and dependent parts; it consists of dusky red, indistinctly defined patches, not raised, and disappearing on pressure; it gives a peculiar mottled appearance to the affected parts. (*b.*) The "mulberry rash" appears about the fifth day on the abdomen and chest, the back of the hands and wrists, and may cover the trunk and limbs. It consists of slightly raised papules, 1–3 mm. in diameter, bright red at first, and disappearing on pressure; in a few days they become crimson or purple from hæmorrhage into the papules, and no longer disappear on pressure. The spots fade away during the second week. In children the rash is much less constant.

The onset of typhus is acute, the temperature is high, and constitutional symptoms are severe.

In rare cases a diffuse red rash like that of scarlet fever appears about the second day, and disappears before the specific typhus eruption shows itself.

Typhoid or Enteric Fever.—The typical "rose rash" appears about the end of the first week (sixth to twelfth day) in the form of rose-pink circular spots, slightly raised but not pointed, 2–4 mm. in diameter, fading under pressure; they are few in number as a rule, and appear first on the abdomen and chest. Each spot lasts three to four days, but successive crops appear day by day until the third or fourth week.

The onset is insidious, the constitutional symptoms variable in severity; diarrhœa is usually present, and the spleen is found to be enlarged.

Careful search in a good light should always be made for the rose-spots in all febrile conditions of obscure origin which have lasted for six or more days.

Acute Rheumatism.—The skin over the affected joints is often red and shining in appearance, but apart from this other cutaneous lesions may be present. These are—

(*a.*) An eruption of small clear vesicles containing sweat (sudamina); these are due merely to the excessive perspiration.

(*b.*) Bright red elevations of the skin, either as papules or larger patches; the latter by their coalescence produce irregular figures, others clear in the centre and form rings. The eruption is known as *Erythema multiforme* and will be again referred to.

(*c.*) Oval or circular elevations of the skin, from a nut to an egg in size, form over the front of the tibiæ and occasionally over the ulnæ. They are usually symmetrical and of a bright or dark red colour, which shades into the healthy skin, and are very tender on pressure. The name *Erythema nodosum* is given to this condition, which is most common in children and females.

(*d.*) *Peliosis rheumatica* is a hæmorrhagic papular eruption, consisting of petechiæ and ecchymoses, occurring chiefly in the lower extremities, but occasionally of wide distribution.

Gout.—The skin over a joint affected with gouty arthritis is dark red in colour, with a tense shining surface and considerable swelling and distension of the surrounding veins: tenderness is extreme, and the swollen skin pits on pressure. Desquamation occurs after subsidence of the inflammation. In chronic gout, whitish deposits, called "tophi," occur immediately beneath the skin in various situations, such as the lobules of the ears, over the knuckles, and round other joints; the skin over them is congested, thin, and shining, and its superficial veins are dilated. The skin often ruptures, and forms a shallow ulcer, which discharges masses of urates ("chalk-stones").

The adjective "gouty" is also applied to various forms of eczema and other diseases of the skin. It does not imply anything peculiar in the form of the disease, but only that it occurs in a patient who suffers or has suffered from the effects of excess of uric acid in the tissues.

(c.) The lesions in the skin are raised above the general surface, and rapidly develop into vesicles or pustules.

1. **Varicella** (**Chicken-pox**) is mostly confined to children. The eruption appears during the first day, usually on the back, and often spreads to other parts of the body; it consists of red circular spots, becoming vesicles in a few hours, and drying up without further change in three to four days. Crusts are formed, which soon drop off, leaving healthy skin. The eruption comes out in successive crops for four to five days; the constitutional symptoms are usually slight.

2. **Variola** or **Small-pox**, and **Varioloid**, or small-pox modified by previous vaccination, sometimes present eruptions on the first or second day quite distinct from the specific eruption of the disease, which appears about the third day. These early eruptions are—(*a.*) a general or partial red rash, closely resembling that of scarlet fever or measles; (*b.*) a hæmorrhagic rash, with a peculiar distribution in the form of a triangle, the apex of which is just below the pubes, the base a transverse line across the abdomen at the umbilicus. The rash consists of small red or purple spots, which, being hæmorrhagic, do not disappear on pressure, but fade after a few days into brown and yellow.

On the third day the typical pocks appear in the form of small papules, usually first on the forehead, where they can be felt as firm "shot-like" bodies in the skin. Similar papules appear on the rest of the face and on the body, they become red, and increase in size until the third day of the eruption (sixth day of the disease), when each presents

at its apex a vesicle filled with clear fluid. The vesicle enlarges, the periphery becoming especially prominent, so as to cause a central depression—umbilication; the clear contents become opaque, and about the fifth day (eighth of the disease) are completely purulent. With the formation of pus the skin surrounding each pock becomes inflamed, red and swollen, and in confluent cases the patient's features may be unrecognisable. The pustules dry up into brown scales, which fall off, leaving a red spot or depressed pit in the skin; if the latter is deep, a permanent record of the disease remains as a dead white depressed scar. The onset is acute, with pains in the back and often vomiting. The temperature rises rapidly, then falls with the appearance of the rash, to rise again with the formation of pus in the pocks. Modified small-pox differs chiefly in the smaller number of pocks, and in their incomplete development, which may be arrested even in the papular, but more frequently in the vesicular stage.

3. **Vaccinia (Cow-pox)** is usually artificially produced on the upper part of the arm, but it is occasionally acquired by accident in unusual situations, and may then cause difficulty in diagnosis. On the second or third day after inoculation a red papule appears; this enlarges, becomes vesicular, and forms a greyish-white circular blister with a depressed centre about the eighth day. The contents become purulent, and a red areola with some swelling surrounds the spot. After a few days it drys up to a brown crust, which falls off during the third week, leaving a permanent depressed scar with a characteristic pitted appearance—foveation.

Class II.—Diseases of the Skin due to Parasites.

The parasites of the skin are both vegetable and animal. The former are entirely fungoid, and include various forms of bacteria and mould fungi; the latter consist chiefly of various species of arthropoda. It is probable that many of the specific fevers already described are really due to parasites of a bacterial nature, but our knowledge is not yet sufficient to justify their being included in this section, where we shall consider those diseases in which parasites have already been demonstrated in the affected skin, and are now generally recognised as the exciting cause of the cutaneous lesions.

(a.) Diseases due to Micrococci.

Erysipelas is inflammation of the skin caused by a specific organism, "*Streptococcus erysipelatosus.*" It presents itself as a red tender spot, which enlarges with swelling of the skin; on pressure the redness disappears, and a pit is left which gradually returns to the previous condition. The affected area spreads at its periphery so as to

invade healthy skin, but often while spreading in one direction it subsides in another. The spreading margin is sharply defined, bright red and raised above the surface; the receding edge gradually fades away into the healthy skin. In severe cases blebs containing serum or pus may form on the inflamed surface, or subcutaneous suppuration may take place with sloughing of the skin. The situation is variable, and is often determined by a wound which is the seat of inoculation. In many cases, however, the face is attacked without any visible wound, such cases being called idiopathic; the swelling of the face may be so great as to render the features unrecognisable. The onset is acute, and the constitutional symptoms severe.

Staphylococcia or **Pyosis** is suppuration of the skin, due to the staphylococcus pyogenes aureus, albus, or citreus. It occurs in various forms according to the site of inoculation.

(α.) **Impetigo Contagiosa** caused by inoculation of a superficial abrasion with the staphylococcus. Each lesion is a discrete vesicle, 3–12 mm. in diameter, the contents of which rapidly become purulent, and dry up into a crust which has the appearance of being "stuck on" to the skin; there is no redness of the surrounding skin, and the crust falls off, leaving a temporary red spot without ulceration or scar. The spots are usually few, but may be numerous, and may coalesce; fresh places are inoculated by scratching, and so keep up the disease for some time. The face, occiput and hands are most frequently affected.

(β.) **Ecthyma** is a large impetigo pustule which has been irritated by rubbing or other cause; the inflammation spreads to the skin beyond the pustule, and a red areola of varying extent is produced.

(γ.) **Furunculus** or **Boil** is due to a deeper inoculation of the staphylococcus, which may gain entrance through the hair follicles or cutaneous glands. It commences as a painful indurated spot, which soon becomes red, raised and pointed; the skin at the apex gives way, and a small slough is discharged. Healing then takes place and a scar is left. Boils may occur on any part of the body. They are especially common in delicate patients.

(δ.) **Carbuncle** differs from a boil in its larger size; it opens by several orifices, and may be compared to a number of confluent boils.

(ϵ.) **Sycosis, Coccogenic Sycosis,** is due to inoculation of the hair follicles of the beard, moustache, or whiskers by staphylococci. The inflammation is more superficial than a boil, and no slough is formed. The disease commences as small papules, each of which is pierced by a hair, the papules soon become pustules, and the pus dries up into a small crust pierced by a hair; the hairs are loosened and the root sheath swollen; their removal leaves small abscess cavities, which

gradually heal up. If the hair papilla is destroyed by the suppuration, permanent baldness remains, also a small depressed scar.

A single patch may be present, or the disease may spread from follicle to follicle until the whole of the hairy part of the face is affected and presents the mass of crusts and pustules from which the name sycosis (fig-like) is derived. The disease never spreads beyond hairy parts.

(b.) Diseases due to Bacilli.

Anthrax, Malignant Pustule, or **Charbon** is due to inoculation of the skin or subcutaneous tissue by the bacillus anthracis. A pimple appears, soon becomes vesicular, bursts and discharges a thin fluid, then dries up into a black gangrenous eschar 5 mm. or more in diameter. The surrounding skin becomes inflamed, and forms a prominent, red, indurated patch of variable size. A ring of small vesicles is found on the swollen patch which surrounds the central black eschar. Constitutional symptoms are usually severe.

Equinia, Glanders, or **Farcy** is due to the inoculation of the skin or mucous membranes by the bacillus mallei. The seat of inoculation becomes inflamed, and the sore ulcerates with thin, foul discharge. After about a week an eruption appears on the face and body; it consists of red papules, which enlarge, indurate, become vesicular or pustular, and rupture to form foul ulcers 10–20 mm. in diameter. Similar lesions are formed in the subcutaneous tissues, intermuscular planes, and on the mucous membranes. Constitutional disturbance is very marked, and of a typhoid character.

Tuberculosis.—Infection of the skin by tubercle bacilli assumes various forms, of which the most common are the following:—

(α.) **Lupus vulgaris** begins as a small red or brownish-red spot; this enlarges, becomes slightly raised and sharply defined, having a peculiar translucent appearance under the epidermis, and a yellowish-brown colour, giving rise to the "apple-jelly" appearance characteristic of lupus nodules. A patch is formed by the coalescence of a number of nodules, and this increases irregularly in size by the formation of fresh nodules in its immediate neighbourhood, which gradually merge into the main patch. Finally cicatrisation occurs, the normal skin being destroyed and replaced by a depressed white scar. This process may occur without ulceration—lupus non exedens—but often as the disease reaches the surface of the skin the epidermis is lost and a shallow ulcer formed, of irregular shape, with raised edges, and covered with dirty greenish crusts. On removal of the crusts, a suppurating base, studded with lupus nodules, is found.

The rate of growth is slow and often intermittent, so that in old-standing cases all stages of the disease are simultaneously present, viz.,

small isolated nodules, raised patches, often scaly on the surface, atrophic scars, and active ulceration, with crusts or discharge.

Lupus is most common on the face, but may be found on any part of the skin. More than one patch may be present, and the mucous membranes are also liable to the disease. The duration of lupus may extend to the whole of the patient's life. The disease is most commonly found in children.

(β.) **Tuberculous ulceration** of the skin usually occurs near the junction of skin and mucous membranes; it is rare and consists of a shallow ulcer of irregular shape, reddish-yellow colour, with a scanty secretion drying up into thin crusts; the ulcer spreads continuously but slowly, shows no sign of healing and, as a rule, is not painful.

(γ.) **Scrofuloderma** is the name given to the lesion of the skin when the latter is involved during the course of a tuberculous inflammation of the subcutaneous tissues or lymphatic glands. The affected skin becomes red and swollen, and penetrated by sinuses. Finally it breaks down to form a shallow, irregular ulcer, with undermined edges and unhealthy granulations secreting thin pus. There is no tendency to heal. The granulations may become hypertrophied, constituting "*Lupus verrucosus.*"

(δ.) **Tuberculosis verrucosa cutis — Verruca necrogenica**, occurs chiefly on the hands of persons who make post-mortem examinations, or otherwise come in contact with tubercular material. It generally begins on the knuckles as a red papule, spreading at the periphery, and gradually becoming horny, so as to resemble a wart; slight purulent discharge is often present, especially when the horny surface has been abraded. There may be only one wart, or several warts may be present on different parts of the hands.

Leprosy—Elephantiasis Græcorum.—This is a chronic disease of the skin, mucous membranes and nerves, due to the bacillus lepræ. The skin lesions appear after a variable period of ill-health as red or brownish spots (*Lepra maculosa*) on the limbs or trunk, of variable size and shape, and slightly swollen; they often fade to pigmented stains or white spots. After an interval, or simultaneously, hard nodules (*Lepra tuberculosa*) appear, mostly on the face and dorsal aspect of the limbs; they vary in size from 5 to 25 mm. in diameter, and when numerous cause great deformity of the features, a condition known as "*Leontiasis.*" The nodules persist indefinitely, but eventually either disappear, leaving a pigment stain, or soften and break through the skin to form indolent ulcers discharging thin pus; the ulcerative process may spread deeply, so as to destroy the tissues of the limbs or open into the joints. In other cases ("*Lepra anæsthetica*") patches of skin become anæsthetic, smooth and glistening, pale or deeply pig-

mented in colour; painless ulceration often takes place and extends deeply, so that fingers or toes may be lost. The course of leprosy is slow, and its duration very prolonged.

Syphilis is a contagious disease, in the lesions of which bacilli have been found, and although proof is still lacking of their causal relationship, the clinical course of the disease renders it extremely probable that such an organism is the actual virus.

The cutaneous manifestations of syphilis present great variety, and may be divided into three groups, viz., *primary lesion* or *hard chancre; secondary lesions*, which are superficial, multiform and numerous; and *tertiary lesions*, which are deep and usually few in number. The boundary-line between secondary and tertiary lesions is not sharply defined, certain forms being classed either as late secondary or early tertiary.

Primary Lesion—Hard Chancre, occurs on some part of the genital organs in about 90 per cent. of all cases. In the remaining 10 per cent. chancres are found on the fingers, face, lips, tonsil, tongue, mamma, navel, anus, or other part of the body. The form of the lesion varies, the commonest being an erosion or fissure, which becomes indurated at its base, does not heal, but remains covered by a scanty non-purulent secretion; another form is that of a papule with a hard base and a desquamating summit; while another is the indolent ulcer with an indurated area round its base—the true *Hunterian chancre*. The induration varies from a thin layer like a piece of parchment, to a mass feeling like a button or a piece of cartilage inserted into the skin. Enlargement of the nearest lymphatic glands soon follows the primary sore, the latter appearing from two to four weeks after exposure to contagion.

Secondary Lesions appear four to six weeks after the primary sore, or six to ten weeks after infection, and may continue from one to two years; they present great variety, and two or more are often present together.

Macular Syphilide.—This is an eruption of rose-coloured spots, which soon become brownish stains. The spots often appear in crops about the seventh week after the primary sore, and affect chiefly the trunk, but sometimes spread to the face and limbs.

Papular Syphilide.—This often appears as the macular eruption is declining; the papules are large or small. The large vary from 3–12 mm. in diameter, are raised and flattened at the top, of a deep red or "raw ham" colour, and are commonly found across the forehead, on the lower part of the face, the trunk, and round the anus and genitals. The small papules are less common, 1–3 mm. in diameter, red or brownish-red in colour, and form irregular groups, each papule in the group being formed round a hair follicle.

Squamous Syphilide, one of the commonest types, is formed by the enlargement or coalescence of papules until raised patches 6–20 mm. in diameter are produced; the surface of each is covered by a scanty layer of dirty-looking scales. On removing the scales, the peculiar brownish-red colour is seen. The rash is widely distributed over the face (especially at the margin of the scalp), trunk and limbs; usually it is most marked on the flexor aspect. Itching is absent or slight. When untreated, the rash may continue for a long time, each spot gradually fading into a brown stain, and similar spots appearing on other portions of skin.

Annular Syphilide.—The lesions are in the form of rings from 10–30 mm. in diameter, with a centre of healthy skin and a raised red or yellowish-red border 2–4 mm. broad, and covered with scales; in many cases neighbouring rings coalesce to form gyrate figures. The face, neck, and trunk are the parts usually affected.

Vesicular Syphilide is a very rare form of rash.

Bullous Syphilide is most common in congenital syphilis in the form of flaccid blebs of various sizes on a red base, and surrounded by a red areola; they contain pus or blood and dry up to a crust covering a spreading ulcer. They may occur soon after birth or during intra-uterine gestation.

Pustular Syphilide is not uncommon in the form of small pustules (*Acneiform syphilide*) on the face, trunk, or limbs; occasionally a few large pustules (*Ecthymatous syphilide*) are present with other lesions.

Rupia is the most severe form of pustular syphilide, and sometimes is classed as a tertiary lesion. It commences as a bulla, which soon becomes filled with pus. This either ruptures or dries up, and a crust is formed under which ulceration spreads; the pus from the ulcer dries up and adds to the thickness of the crust from below, and as each successive addition is larger in area than the previous one, a conical "limpet-shell" crust of great thickness is produced, removal of which exposes the ulcerated surface.

Pigmentary Syphilide is rare, and is chiefly found on the neck in women as yellowish-brown spots of round or oval shape, often confluent, with the patches separated by abnormally white skin, so that the neck has a peculiar mottled look. This lesion must not be confounded with the brown stains left on the skin by the fading of any form of syphilitic eruption.

Tertiary Lesions rarely occur before six months, and may never appear at all. In addition to rupia they include—

Nodular Syphilide—Cutaneous Gummata.—These are elevations of the skin, from a pea to a bean in size, of a livid red, "coppery," or "raw ham" colour, and often breaking down into serpiginous ulcers; they

occur on the face, trunk and limbs, and are often grouped in characteristic crescentic or horse-shoe patches. When on the nose they closely simulate lupus.

Subcutaneous Gummata are firm, well-defined nodules, deeply embedded in the subcutaneous tissues. As they enlarge, the skin over them becomes adherent, and finally breaks down to discharge purulent contents, and leaves a deep crater-like ulcer with well-defined, "punched out" edges. Gummata occur chiefly on the scalp, face, clavicles and bones of the limbs.

The mucous membranes of the mouth and pharynx, and genital and anal orifices are affected by syphilis, but the appearance of the lesions is modified by the warmth and moisture. Lesions of those parts of the skin where similar conditions of warmth and moisture prevail (as, for instance, between the toes, in the fold of the nates, or in the axilla), present the same appearance, which is that of raised flat patches, white, red, or brownish-red in colour, and varying from the size of papules to 10 or 15 mm. in diameter. The epidermis, softened by the moisture, is soon rubbed off, and a secreting surface left, covered with thick contagious pus. The names *mucous tubercle*, *mucous plaque*, or *condyloma* are given to these lesions.

The hair may fall out, and the nails show various alterations of nutrition. Visceral disease in various forms occurs in the late stages.

Congenital Syphilis differs from the acquired form in the absence of primary lesion, and also in the greater predominance of lesions of the mucous membrane of the nose, causing "snuffles." The skin eruptions are of macular, papular, squamous, or bullous type. The last-named is most frequently found on the hands and feet; the other varieties usually commence round the buttocks, and may spread extensively. Mucous tubercles round the mouth and anus are often present, and other lesions affect the eyes, teeth, epiphyses of bones, periosteum, or viscera. Children with congenital syphilis are often born dead, or die soon after birth, but many cases are apparently healthy until three to four weeks old.

The diagnosis of syphilitic lesions rests upon the peculiar reddish-brown, raw ham, or coppery colour; the simultaneous presence of several types of eruption; the affection of parts of the body not usually attacked by non-syphilitic eruptions; the absence of itching; the presence of marked pigmented stains after the active stage has passed away; the serpiginous character of the ulcerations produced; the presence of lesions of the mucous membranes of a syphilitic character; the loss of hair; the presence of a cicatrix of the primary lesion; general enlargement of lymphatic glands; the existence of evident cachexia or of syphilitic disease of the viscera.

(c.) Diseases due to Mycelial Fungi.

Actinomycosis of the skin is rare, and usually secondary to affection of the deeper tissues: it is due to growth of the ray fungus setting up suppurative inflammation of the tissues.

The involved skin becomes reddened and thinned, and gives way at one or more places, usually in some part of the face or neck, and a purulent fluid is discharged containing yellow granules 1–3 mm. in diameter. On microscopical examination these granules are found to consist of the fungus.

Favus—Tinea favosa, is rare in England, but common in Scotland and on the Continent. The scalp is usually affected, but any part of the body may be the seat of the disease, which appears first as a reddish itching patch, soon presenting minute circular spots of a sulphur-yellow colour; these project and enlarge, the centre is depressed and pierced by a hair, so that a yellow scutullum or favus cup is formed from 10–12 mm. in diameter.

Neighbouring cups become confluent to form a continuous thick yellow crust, with an irregular honeycombed surface, and emitting a peculiar mouse-like odour. Isolated cups are usually found at the margins of the crust. On removal of the latter, the skin presents a reddened appearance, is slightly depressed, and sometimes scaly; in late stages the skin becomes thin and white and devoid of hair.

The hairs are loose, dry and brittle, they fall out, the follicle becomes atrophied and permanent baldness may result. On non-hairy parts the favus cups present the same appearance, and circular rings of red papules are often found.

The nails may also be affected by favus. The fungus causing the disease is the *Achorion Schönleini*. It can be found by soaking affected hairs or yellow crusts in liquor potassæ, and pressing them out beneath a cover-glass. It consists of a branching mycelium, the threads of which are made up of ovoid segments 3–4 μ in diameter, placed end to end; nucleated spores are also present of comparatively large size, 7 μ in diameter, and either in rows or in groups.

Ringworm.—Tinea tonsurans, ringworm of the scalp—**Tinea circinata**, ringworm of the body—**Tinea sycosis vel T. barbæ**, ringworm of the beard.—The hairy scalp is most commonly affected. The disease commences as a red papule round a hair; this spreads at the periphery to form a red, slightly raised patch covered with greyish-white scales, through which project the stumps of broken hairs; the patch enlarges up to 2–5 cm. in diameter, and may coalesce with others to form an irregular area, or may involve the whole scalp. The hairs are dull in colour, very brittle, and broken off about 4 mm. from the skin; the stumps are often bent or twisted, and the extremities frayed out like a brush.

Examined under the microscope in liquor potassæ, the hair shafts are seen to be infiltrated with spores and mycelial threads of the *Trichophyton tonsurans;* the former are about half the size of favus spores, and the latter are less jointed and run longitudinally in the hair.

FIG. 62.—Hair with Favus fungus. A, A, chains of spores projecting beyond the edges of the hair; B, spores between the fibres of the hair; C, D, broken-up root end of the hair, with masses of spores between the laminæ. (*Küchenmeister.*)

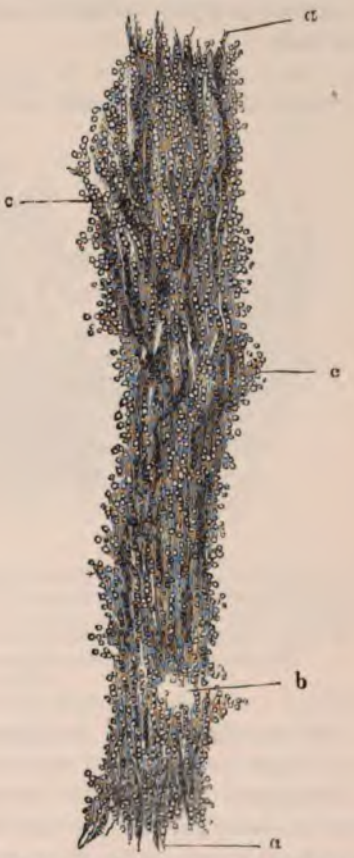


FIG. 63.—Hair from a case of Tinea tonsurans, loaded with spores. *a, a*, broken ends of hair; *b*, rupture of longitudinal fibres; *c, c*, ragged edges of hair. (*M'Call Anderson.*)

In some cases the condition known as *kerion* is produced, in which there is suppuration of the hair follicles in the affected area, which forms a red, raised patch, soft and boggy to the touch, and discharging

pus from several points; the hair follicles are usually destroyed by the suppurative process and permanent baldness results.

Ringworm of the body appears as a circular patch 10–25 mm. or more in diameter, with well-defined slightly raised edges of a reddish colour and covered with branny scales; as the periphery spreads, the centre gradually fades and clears up, so that a ring is formed which may coalesce with neighbouring rings to form gyrate figures covering a large area.

Scrapings of the epithelium show the same fungus as ringworm of the scalp.

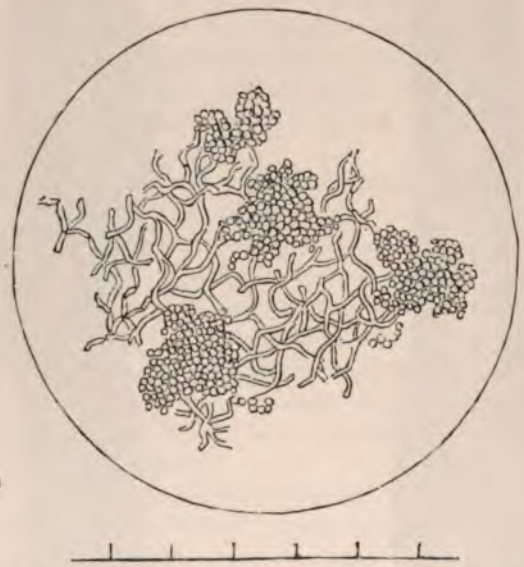


FIG. 64.—Shows the clusters of spores and the tubes of the Microsporon Furfur. (*M'Call Anderson.*)

The so-called *Eczema marginatum* is ringworm affecting the fork and neighbouring parts of the trunk and thighs.

Ringworm of the beard is the result of inoculation of the Tricophyton into the hairy parts of the face; it begins as a red itching spot, which may form a scaly patch or ring, but in most cases a suppurative inflammation is set up in the hair follicles, which become swollen, forming tender, red, prominent nodules, in which points of suppuration are seen with loosened hairs; the follicles may be destroyed and bald patches result.

Tinea versicolor or **Chloasma** is found almost entirely on the trunk as yellowish-brown or buff patches of various shapes and sizes. These are scarcely raised above the surface and very slightly scaly at

the margin; neighbouring spots enlarge and coalesce to form large areas, which may cover the greater part of the trunk.

A scraping of the epithelial scales from the edge of a patch, when examined in liquor potassæ under the microscope, shows a network of mycelium studded with collections of round spores, the *Microsporon furfur*.

Erythrasma, a somewhat rare disease, similar to Tinea versicolor, but of darker colour, occurs in the folds of the axilla, or the fork and neighbouring parts; it is due to a small fungus, the *Microsporon minutissimum*.

Alopecia areata appears in the form of localised bald spots on normally hairy parts, most commonly the scalp; they vary in number,

FIG. 65.—A burrow containing a female acarus with the head directed to the blind end of the burrow. Within the acarus is an egg. Behind the acarus and in a row one after the other, with their long axis placed transversely to the long axis of the burrow, there are ten ova in various stages of development. Between the ova are black irregularly-shaped fæcal masses. (*After Neumann.*)

size, and shape, but all present a smooth, glistening surface. Around the border of the patch scattered among the healthy hairs are found short stumps of hair-shafts, thicker at their free extremity, and often compared to a point of exclamation (!). The patches spread peripherally, and during recovery the broken hairs are no longer found, but a fine downy growth of hair appears on the smooth glistening skin.

The etiology of the disease is still doubtful; probably both neurotic and parasitic cases are at present included under the same name.

(d.) Diseases due to Animal Parasites.

Scabies.—Itch is a contagious disease characterised by intense itching of the skin from the presence of the *Acarus Scabiei*, the so-called itch insect. The lesions in the skin are multiform, and are either due

to the actual presence of the parasite or to the friction or scratching of the skin of the patient. The characteristic lesion is the *burrow* (see Fig. 65), a tunnel formed in the skin by the female acarus, and in which the eggs are laid and hatched; on the surface it appears as a white or dark line (usually the latter, from particles adherent to the slightly roughened epidermis forming the roof of the burrow), 3–17 mm. in length, and often more or less curved. At the deeper end the parasite is to be found, while the eggs in various stages of development occupy the remainder of the burrow. The parts most frequently affected are the interdigital webs and dorsal aspect of the fingers, flexor surface of the wrist, penis, and neighbouring parts. The para-

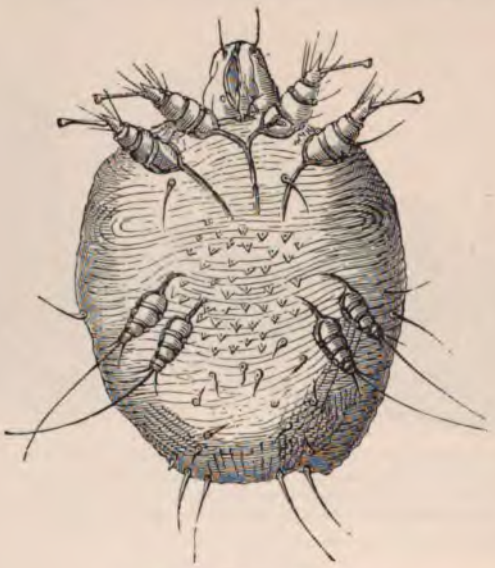


FIG. 66.—Female Acarus.

site may be obtained for examination by carefully scraping the epidermis over the deeper end of a burrow with a pin until the roof is broken through, the acarus can then be picked out by the pin, to the point of which it usually clings. It forms a minute, oval white speck, .3–.4 mm. in length; under the microscope it is seen to possess four pairs of legs, the two anterior pairs having suckers at the extremities, the two posterior ending in bristles or *setæ*. The male is smaller than the female, and the fourth pair of legs are also furnished with suckers; it does not burrow, but wanders over the surface of the skin.

The site of a burrow is often marked by a papule, vesicle, or pustule, the result of inflammation set up by the presence of the parasite.

The lesions due to scratching consist of linear wheals, excoriated papules, vesicles and pustules, with crusts of dried blood, pus, or serum. The eruption is chiefly on the front of the body and limbs, and is almost confined to the area bounded above by a transverse line across the chest and arms at the level of the nipples, and below by a transverse line at the level of the knees. In patients who sit much on hard seats, the region of the buttocks is much affected, and so are the lines of pressure from belts or tight clothing.

The characteristic feature of scabies is the variety of the inflammatory lesions and their distribution, while the presence of a burrow is conclusive.

Pediculosis—Phthiriasis, occurs in three forms, according to the three species of lice which are found as human parasites. These are named, from their habitat, *Pediculus capitis*, *Pediculus corporis* vel *vestimentorum*, and *Pediculus pubis*.

Pediculus capitis.—The head-louse lives and breeds in the hair of the scalp; it is about 2 mm. long, and half as much broad, of a dirty white colour, and consists of a triangular head, a thorax, to which are attached six legs, and an abdomen, which comprises the bulk of the body of the animal.

FIG. 67.—Pediculus capitis. (*M'Call Anderson.*)

The lice are found wandering about the roots of the hairs, while the ova ("*nits*") are found as small white specks glued laterally to the hair shafts.

The presence of the parasites excites intense itching and scratching, especially in the occipital region. Excoriations are formed, which become inoculated by pus cocci, and pustules are produced. This process spreads until the back of the head becomes covered with a thick mass of matted hair, crusts and dried secretions, concealing a suppurating surface. To this condition the name *Eczema Impetiginodes* is often applied. The sub-occipital glands are enlarged and may suppurate.

Pediculus corporis, or body-louse, is larger than the head-louse, and wanders over the surface of the trunk for the purpose of feeding; it inhabits the clothes of old and dirty people, especially about the neck and shoulders.

The characteristic lesion is a minute hæmorrhagic speck, just visible to the naked eye, and not raised above the surface of the skin. The itching is most intense, and the skin of the affected area is covered with a typical scratch eruption, made up of linear wheals and excoriations, small papules with a dried blood crust on the summit, occasional pustules and, in chronic cases, much pigmentation of the skin.

Pediculus pubis.—The crab-louse is broader, smaller and flatter

than the others. It is found in the pubic hair and occasionally on the hair of the face. Its presence leads to great irritation and scratching, with similar results to those produced by the Pediculus capitis on the head.

Pulex irritans, the common flea, produces a slightly raised red spot, with a central puncture, often marked by a speck of blood, which persists as a petechia after the hyperæmic papule has disappeared.

Cimex lectularius, the bed-bug, produces a raised red spot, similar to but larger than that caused by a flea, and more persistent and irritating; a purpuric spot remains for some time and undergoes the usual changes in colour.

FIG. 68.—Pediculus corporis, female. (*M'Call Anderson.*)

FIG. 69.—Pediculus pubis. (*M'Call Anderson.*)

FIG. 70.—Acarus folliculorum, fully matured specimen, dorsal view. (*Nayler.*)

Leptus autumnalis, the harvest-bug, bores into the skin and causes a papular eruption, which itches and leads to scratching and the consequences thereof. The lower parts of the legs are usually first affected.

Gnats, mosquitoes, bees, wasps and some **caterpillars** cause papules and wheals, with much itching and irritation, and sometimes considerable swelling.

Acarus folliculorum is a parasite not unfrequently found in the sebaceous glands; it is of elongated shape, one-sixth to one-third mm. in length, and consists of a head, a thorax, with four pairs of rudimentary legs, and a tapering abdomen, which forms about one-half

of the entire length of the animal. It gives rise to no symptoms in man.

Elephantiasis—Elephantiasis Arabum, consists in great hypertrophy of the skin and subcutaneous tissues from obstruction of lymphatics by parasites (*Filiaria sanguinis hominis*) or other causes. The affected part, often the leg (*Barbadoes leg*) or scrotum, is enormously swollen, but does not pit on pressure. The skin is thickened, hard, smooth, or irregular from dilated lymphatic vessels. The latter often rupture and discharge clear or milky fluid; pigmentation is often present and varies in character.

Class III.—Local Diseases of the Skin not due to Cutaneous Parasites.

(a.) The changes in the skin are alterations of colour or consistence, or both combined. There is no elevation of the affected surface, no formation of vesicles, pustules, or secondary lesions.

Erythema simplex is a patchy redness of the skin from the action of some irritant, such as friction, heat, the rays of the sun, or chemical irritants.

Erythema intertrigo is a redness of the skin due to friction of contiguous surfaces, such as the groins and thighs of children, or beneath the mammæ in obese females.

Erythema læve is a more or less diffuse redness, often seen on skin affected with œdema.

Erythema pernio—Chilblains, are red patches on the fingers, toes, or other exposed parts of the body where the circulation is feeble, caused by exposure to cold, and especially occurring in persons of feeble circulation. If exposed to friction, inflammation and ulceration of the skin may take place.

Erythema paratrimma is the redness of the skin which is exposed to pressure when patients are confined to bed; it is especially liable to form when there is any disease of the spinal cord; if not relieved, ulceration takes place, and the well-known *bedsore* (*Decubitus*) is formed.

Purpura is a name given to hæmorrhage into the skin from various diseases; it sometimes appears without obvious cause, and is then described as a separate disease; the skin presents numerous purple spots of all sizes, from petechiæ up to large irregular patches; these do not disappear on pressure, and undergo the usual changes in colour seen in a bruise. The legs are usually first affected, but in severe cases, *Purpura hæmorrhagica*, the greater part of the body may

be involved, and also the mucous membranes, so that hæmorrhage may occur from nose, mouth, respiratory tract, stomach, or rectum.

Cutaneous nævus is a vascular growth in the skin made up of dilated capillaries or veins; it is found as a red or purple patch of variable size and shape, often on the face as a "port-wine stain" or "mother's mark." Pressure empties the blood-vessels, which gradually refill when the pressure is removed. Nævi are often present at birth, or appear soon after, but may occasionally appear in adults; they may either remain stationary or gradually increase in size. Dilated blood-vessels are also found on the face in *Acne rosacea* (*q.v.*), and may be the only lesion in the early stages.

Lentigo, Ephelides, Freckles, are small spots of brown or yellow pigment, occurring on those parts of the body exposed to the light, most commonly the face and backs of the hands.

Albinismus is a congenital absence of pigment from the body; when complete, the skin is white or pinkish, the hair white, and the eye appears pink owing to absence of pigment in the iris and choroid.

Leucoderma—Vitiligo, consists in a deficiency of pigment over limited areas of skin, with excessive pigmentation in the parts immediately surrounding the diseased areas; it appears in the form of white patches, 1–10 cm. or more in diameter, of irregular shape, but having well-defined convex borders, which sharply contrast with the deeper colour of the surrounding skin. The patches enlarge peripherally, coalesce, and may spread over nearly all the body; the hair on the affected skin becomes white; recovery rarely takes place.

Scleroderma—Hide-bound Disease, occurs in two distinct forms:—

(*α.*) **Diffuse scleroderma** is very rare, and begins about the face or neck as a hardening of the skin without change of colour; there is usually no swelling of the skin; the affected area does not pit on pressure, and the skin cannot be pinched up from the subcutaneous tissues. The movements of the joints are stiff, and finally completely lost. In other cases œdema is an early symptom, and later gives place to induration and shrinking. The affected skin after some time often shows reddish patches, and yellow or brown pigmentation. The greater part of the body may be affected, and the disease may last for years.

(*β.*) **Circumscribed Scleroderma—Morphœa—Keloid of Addison,** presents itself as an irregular-shaped patch of a pale yellow or ivory colour surrounded by a zone of purplish hyperæmia; the patch is neither raised nor depressed; the skin feels like parchment or leather, is smooth, dry, and can often be pinched up. Several patches are often present at once, and are usually 3–8 cm. in diameter; they persist for years.

Striæ atrophicæ—Lineæ albicantes, are white lines on the skin of

the abdomen, due to localised atrophy of the skin following previous over-stretching, as in pregnancy or ascites; other parts of the body are sometimes affected with similar or more extensive atrophic patches.

Glossy Skin is a condition usually seen in the hands as the result of injury or disease of the nerves supplying the affected part. The skin is thin, smooth, glossy and without furrows, and redder than normal, or mottled red and white. Neuralgic pain is often present.

(b.) The lesions in the skin are localised elevations of the surface; vesicles, pustules or secondary lesions are only produced as the result of scratching or other irritation.

Callositas—Tylosis is a localised thickening of the epidermis, usually found on the palms and soles, and due to intermittent pressure from the use of tools, from much walking, or similar cause.

Clavus—Corn, is a similar localised thickening of epidermis, but it grows downwards into the skin, causing atrophy of the papillæ and the formation of a pit, which is filled by the epidermic plug; pressure on the elevated horny surface causes acute pain. The toes are the parts chiefly affected; when the corn is between adjacent surfaces the epidermis is macerated by the moisture and soft corns are produced.

Verruca—Wart, is a small projecting growth composed of one or more hypertrophied papillæ covered by a thick layer of horny epidermis; warts may be pointed or flattened, single or in enormous numbers. When recent they are pink in colour; when old, yellowish brown. The hands of children are the most common sites.

Venereal Warts—Condylomata, occur on the perineum, penis, vulva, or other moist situations, as the result of irritating discharges; they are pink or red in colour, and usually covered by a foul white secretion.

Nævus pigmentosus—Pigmentary Mole, is a small collection of brown or black pigment in the skin, which is usually more or less elevated above the surface; many are covered by hair (*Nævus pilosus*). Moles vary much in size and number; the face, neck and back are the most common sites.

Xanthoma—Xanthelasma, consists of small buff-coloured, slightly raised spots on the skin. The commonest situation is the upper eyelid near the inner canthus. They are formed in association with "sick headache" and with jaundice.

Keloid—Keloid of Alibert, is a nodular growth of fibrous tissue occurring on scars (so-called false keloid) or spontaneously. It forms smooth, white or pink, elevated masses of various shapes, the most common being a flat rod with lateral processes, situated over the sternum. Contraction of the new-formed tissue may cause deformity of the surrounding skin.

Melanotic Sarcoma appears as one or more raised, brown, or bluish-black spots, which enlarge to form distinct tumours. They often originate from a pigmented mole.

Molluscum fibrosum is a soft growth projecting from the surface of the skin, often pedunculated, and varying in size from a pin's head to an orange; the skin of the tumour is normal in character and often flaccid and wrinkled. The tumours may be present in very large numbers.

Molluscum contagiosum consists of small growths on the skin, 2–12 mm. in diameter, sessile or pedunculated, of a yellowish colour and firm consistence. The top is usually pitted with a small central hole, from which a little milky semi-fluid material can often be squeezed. Two or three may be present on the face, hands, arms, mammæ, or genitals, or the growths may be scattered all over the body.

Prurigo is a rare disease, which usually begins in early infancy and persists through life. It must be carefully distinguished from *pruritus*, *i.e.*, the symptom of itching, which is common in many forms of skin disease. In prurigo, the lesion consists of minute papules, more easily felt than seen, as they are of the colour of the skin. They chiefly occur on the extensor surfaces, and are attended by intense itching and scratching, with the usual effects of the latter upon the skin.

Urticaria—Nettlerash, is characterised by the evanescent character of the eruption and by the intense itching of the skin. The lesion consists of wheals, slightly raised red or white elevations above the surface, of various shapes and sizes, but always very broad in proportion to their height; they may disappear within an hour, or may last several days; successive crops of wheals may continue the disease for an indefinite time.

In children, persistent papules (*Lichen urticatus*) are often left after the wheals have disappeared. Disturbance of the alimentary canal by ingesta is often associated with the disease.

Erythema multiforme occurs, as already mentioned, with rheumatic symptoms, but also independently. The lesion consist of red papules (*Erythema papulatum*) of varying size; these may remain discrete, or run together to form slightly elevated red patches of irregular shape. The swelling and redness often disappear from the centre of the patch, so that a ring (*Erythema annulare*) is formed, and, by the coalescence of neighbouring rings, gyrate figures (*Erythema gyratum*) are produced.

(c.) There is persistent scaly desquamation from the skin, either general or local.

Ichthyosis—Xeroderma, is a congenital condition of the skin due to thickening of the epidermis and deficient secretion of the cutaneous glands.

In the mild forms (**Xeroderma**) the skin is dry, rough, and looks

dirty, especially over the elbows, knees and extensor surfaces of the limbs. In more advanced cases the dry skin is covered by thin polygonal scales of epidermis. On their removal the skin beneath is seen to be of normal colour. The worst cases (**Ichthyosis hystrix**) are characterised by the formation of horn-like polygonal masses of epidermis, 10–15 mm. in height, and of a dark greenish colour. The intermediate skin is generally xerodermatous, but may be healthy.

Lichen scrofulosorum is an uncommon disease which occurs in scrofulous patients; it consists of minute papules of a red or pale colour, each papule being covered by a minute scale. The trunk is the part affected.

Lichen planus is a somewhat rare disease, characterised by discrete papules, 2–4 mm. in diameter, of a bluish-red colour and angular outline. The apex is smooth and flattened, and presents a central depression; the papules, when once formed, do not enlarge, but fresh papules appear between those already present until confluent patches are produced of various size and shape; these are of a purplish colour, raised, firm and covered with scales. The front of the wrist and forearm and inner aspect of the knees are chiefly affected, but it may appear on any part of the body.

Psoriasis is a common disease, which usually commences over the patellæ and olecranon processes as small, raised, red spots covered by silvery grey adherent scales. On completely removing the scales, the red surface exhibits to the lens a number of bright red dots. The patches increase in size, and coalesce to form larger patches of varied outline. Different names are applied to the different appearances presented, such as *punctata*, when the spots are small; *guttata*, when they are about 5–6 mm. in diameter, and look like drops of mortar on the skin; *nummularis*, when larger; *circinata* and *gyrata*, when rings or gyrate figures are formed by involution of the central part of each patch and coalescence of neighbouring rings; *universalis*, when the whole of the body is involved. The eruption is dry throughout the whole course of the disease.

The extensor surfaces of the limbs are the most common sites; the scalp is often involved, then the trunk, less frequently the face and flexor surfaces of the limbs; the eruption is usually symmetrical, and may spread over the whole of the skin. The general health is hardly affected, and recurrence commonly takes place.

Pityriasis rubra, Exfoliative dermatitis, is rare, and begins as a red patch on the chest or arms; this rapidly spreads, and other patches form and coalesce, so that in a few days the whole surface of the body is involved, and presents a surface of bright red colour, covered by thin scales, small on the face, but forming large flakes

on the trunk and limbs. The scales are shed copiously. There is often burning and tingling, but not much itching. The skin is very little thickened, discharge is usually absent, and if present does not stiffen linen. The patient is often in good health, but constitutional disturbance may be severe, and even fatal in elderly people, among whom an epidemic form of the disease has recently been described.

Seborrhœa and **Seborrhœic dermatitis.**

(*α*) **Seborrhœa sicca** affects chiefly the scalp and other hairy parts; it consists in accumulation of the dried fatty secretions of the cutaneous glands to form dried scales (*scurf* or *dandriff*) covering the skin at the roots of the hairs. On removal of the scales, the skin is white and apparently normal. The disease leads to atrophy of the hair and baldness.

(*β*) **Seborrhœa oleasa** is seen chiefly on the face as an oily secretion, giving a shining, greasy look to a skin which is usually dirty from adherent dust. It also occurs on hairy parts, when a greasy crust of fat and epithelium is formed, instead of the dry scales of seborrhœa sicca.

(*γ*) **Seborrhœic dermatitis** is an inflammation of the skin due to, or associated with, the disorder of the cutaneous glands causing seborrhœa; the lesions of the skin closely resemble those of the less acute forms of eczema (*q.v.*), less frequently those of psoriasis, while a peculiar form affecting the trunk is known as *Lichen circinatus*, and consists of red spots spreading peripherally, and healing in the centre to form rings; the central portion is of a light yellow-brown colour, and the red borders are scaly. Neighbouring rings coalesce to form gyrate figures, which may cover the greater part of the trunk; the limbs are not involved.

In the various forms of seborrhœic dermatitis the scales and crusts are greasy, and seborrhœa is present on the scalp or other parts.

Lupus erythematosus is not very common; it occurs chiefly on the face, but is also found on the scalp and other parts of the body; the cheeks and bridge of the nose are often affected together, giving the so-called "butterfly" patch. It commences as a red spot, covered by a yellow, adherent, greasy scale; if this be carefully removed, a process from its under surface is seen to project, like a plug, into the orifice of a sebaceous gland. The spots spread peripherally, and coalesce to form red patches of varied size covered with similar scales and plugs; the margin of the patch is sharply defined and slightly raised above the surface. After an indefinite time the centre of the patch becomes depressed, scales no longer form, and a white superficial scar is left; when hairy parts are involved, permanent baldness results. Ulceration never takes place. The duration is very prolonged, progress

is slow and often intermittent, periods of long quiescence being interrupted by acute exacerbations and rapid extension of the disease.

(d.) Vesicles or bullæ are present on the skin at some stage of the disease; either absorption, or rupture and discharge of contents takes place, with formation of crusts, or desquamation of epithelium.

Miliaria, sudamina, are minute vesicles, filled with clear, slightly acid fluid. They are due to the accumulation of sweat beneath the epidermis, as a result of profuse perspiration. They may become inflamed and form papules, the so-called *red gum* or *strophulus* of infants.

Cheiro-pompholyx.—A disease limited to the hands and feet; it consists of vesicles situated beneath the horny epidermis, through which they appear as small translucent spots; when numerous and closely set, they coalesce into irregular bullæ. The skin is not reddened, but there is often burning or tingling. After seven to ten days the contents are absorbed, and the epidermis peels off, leaving normal skin beneath.

Erythema iris—Herpes iris, is closely allied to Erythema multiforme, and occurs as a red papule, soon becoming a vesicle; this enlarges, the centre is absorbed, and a vesicular ring is left surrounded by a red areola; in other cases a ring is absorbed leaving a central vesicle surrounded by a depressed purplish-red ring; this is again surrounded by a white vesicular ring, and finally a red areola. Occasionally the number of rings may be increased or irregular figures formed by coalescence.

Herpes facialis occurs in association with acute pneumonia, bronchitis and ordinary catarrh, as one or more groups of vesicles on an inflamed base. The contents dry up into a scab, which soon falls off. The lips, nose and adjacent parts are usually affected.

Herpes preputialis occurs on the prepuce and neighbouring parts, and occasionally on the vulva in females. One or more vesicles are found on an inflamed base; rupture usually occurs and shallow ulcers are formed, which must be distinguished from venereal sores.

Herpes zoster—Zona—Shingles, consists of an eruption of papules, which rapidly become vesicles on an inflamed base; the vesicles are arranged in groups, which correspond in distribution to the branches of some cutaneous nerve; one of the intercostal nerves is most commonly affected, the eruption forming a girdle (*zona*) round half the trunk from spine to sternum. The branches of the fifth cranial nerve or the nerves of the limbs may also be involved. There is often neuralgic pain along the course of the affected nerve, which may persist for some time. In most cases, if not in all, the lesion is a trophic one, due to

disease in the ganglion on a posterior root, or its homologue in the case of the cranial nerves.

Pemphigus presents an eruption of bullæ which gradually increase to 3 cm. or more in diameter; each is surrounded by a red areola. Any part of the body may be affected; the number of bullæ varies, and they come out in successive crops. Each crop lasts a few days, when the fluid is absorbed or discharged, and the epidermic covering dries and falls off leaving a red stain.

Pemphigus foliaceus is a rare and fatal form, in which the whole body may be involved. The eruption consists of flaccid bullæ, which rupture and form foliaceous crusts covering a raw inflamed surface.

Dermatitis herpetiformis is a rare disease, which affects chiefly the limbs and abdomen; it presents itself as red, flat patches with a raised margin and a depressed livid centre; these gradually enlarge, and when 2–3 cm. in diameter, vesicles of various size develop on the raised red margin; bullæ also form, both on the raised patches and independently. Fresh patches form during the course of the disease, so that all stages are found present at the same time.

Eczema is the most common of all diseases of the skin, and manifests itself by a great variety of lesions, according to the stage of inflammation reached in each particular case, and the nature and extent of the secondary lesions. The essential feature is the superficial inflammation of the skin. This shows itself in various ways, so that different names are applied to the different stages.

(*a.*) **Eczema erythematosum** usually occurs on the face as diffuse red patches, with much heat and some swelling of the cellular tissue, the superficial epidermis is shed as fine scales, and the inflammation either subsides or passes on to a more intense degree.

(*b.*) **Eczema papulosum—Lichen simplex**, is characterised by the formation of red papules on the trunk, limbs, or other parts; the papules vary in number, and may coalesce to form raised red patches; vesicles may be formed and discharge serum which stiffens linen, or the epidermis be shed in the form of scales. There is much itching and irritation, which often leads to scratching and excoriations.

(*c.*) **Eczema vesiculosum** occurs most frequently on the flexor surfaces of joints, behind the ears, or anywhere where the skin is thin; it appears as a red patch, which burns or itches, and soon presents a number of minute vesicles containing clear serum; the vesicles rupture and the serum forms a discharge, which is often copious, and stiffens linen on drying; if scanty, the discharge dries up to form yellow crusts, on removal of which the red, moist, exuding surface is seen.

This, the classical type of eczema, may develop from either of the preceding forms.

(*d.*) **Eczema rubrum** is a still more intense degree of inflammation, and often affects large tracts of skin, especially on the lower extremities; the inflamed surface is bright red and moist, discharging large quantities of serum, which dries up into yellow crusts, not infrequently mixed with a little blood from rupture of the distended capillaries.

(*e.*) **Eczema squamosum** is a much less intense inflammation, and is often a later stage of any of the more acute conditions; it occurs as irregular patches of a red colour covered by scales of epidermis. On pinching up the skin, it is felt to be thickened and indurated.

(*f.*) **Eczema pustulosum—Eczema impetiginodes,** is due to secondary inoculation of the inflamed skin by pus cocci, so that pustules form with greenish crusts of dried pus; this is not uncommon, and is distinguished from Impetigo contagiosa by the fact that the pustules in the latter disease occur on healthy skin, and not on an already inflamed surface.

(*g.*) **Eczema rimosum** occurs most frequently on the hands of people who work in irritating material, such as sugar, chemicals, or soap and water; there is much thickening of the epidermis, which interferes with the movements of the joints, and cracks take place in the lines of flexure, exposing the red surface of the papillæ and forming painful fissures.

In a typical case of eczema, several of these varieties are present together, the skin presenting papules, vesicles and red patches, crusts and weeping surfaces, or thickened skin covered with scales, according to the stage and degree of the inflammation.

Eczema may persist for years, but usually by successive outbreaks of a more or less acute character, each of which leaves behind an increase of inflammatory infiltration, so that the affected skin becomes thick and hard in proportion to the chronicity of the disease.

(e.) The affected skin presents suppuration of the sebaceous glands, combined with other lesions which vary in character.

Acne vulgaris commences by a blocking of the ducts of the sebaceous glands and retention of the secretion; this gives rise to a prominent white papule, the opening of which presents a black dot, due to accumulated dirt in the orifice of the duct. Such a papule is called a "*Comedo,*" and the retained secretion can be expelled by pressure. Comedones are most commonly found on the face, and may persist indefinitely; usually, however, suppuration takes place, the papule increases in size, becomes red and painful, the summit soon presents a

yellow spot due to the formation of pus, the minute abscess ruptures, the contents are evacuated, and healing takes place either with or without scar according to the depth to which the suppuration has extended.

The face, neck, shoulders and chest of young adults are the parts particularly affected.

Acne rosacea—Gutta rosea, is a condition of hypertrophy of the vessels of the face often associated with disorder of the alimentary canal, abuse of alcoholic stimulants, or uterine derangements.

The nose is usually first affected, and is red, with distinct tortuous blood-vessels on the affected part; at a later stage nodular thickening of the nose (so-called "grog blossoms") present themselves, and may increase in size so as to cause considerable deformity. After the nose, the condition spreads to the cheeks, chin and forehead. Combined with the vascular hypertrophy there is usually inflammation of the sebaceous glands of the affected area, leading to suppuration and the formation of pustules.

(f.) The skin presents irregular ulceration, with indurated edges, spreading deeply, and showing no signs of healing.

Epithelioma frequently affects the skin of the lower lip or genitals, but may occur on any part of the body. It begins as a small hard wart or nodule, which gradually enlarges and becomes adherent to the deeper tissues. The surface ulcerates, and a constantly spreading ulcer is produced, with raised, indurated, everted, or undermined edges, and an irregular base, with more or less foul discharge. The disease spreads so as to destroy the whole structure of the affected part. The nearest lymphatic glands are usually enlarged and indurated.

Rodent ulcer occurs chiefly on the eyelid, nose, or cheek as a soft, wart-like growth, which breaks down in the centre to form an irregular ulcer, with hard sinuous edges, not everted, undermined, or nodular, and of a yellowish-red colour. The surface presents few granulations and little discharge. Pain is absent or slight, duration prolonged, and the neighbouring lymphatic glands are not affected.

(g.) There is gangrene of the skin.

Acute decubitus—Bedsore, is gangrene of that part of the skin which is compressed by the weight of the body when the patient is confined to bed from disease or injury, more especially of the central nervous system. The gangrene is moist in character.

Cancrum oris is gangrene of the substance of the cheek, commencing in the mouth and spreading to the skin. It occurs in weakly children, especially after various specific fevers.

Noma is a similar condition, affecting the external genital organs of female children.

Senile gangrene occurs chiefly on the lower extremities of old people; it commences as a black spot on the toes or foot, and spreads to a variable extent, the affected area becoming dried up, shrivelled-looking and black.

Raynaud's Disease is a rare disease, affecting the extremities in a symmetrical manner; the fingers or toes are cold and numb, then blue and congested, and finally black gangrene occurs, and the affected parts are separated in the usual way.

Drug Eruptions.—(*Abridged from Dr. R. Crocker, "Diseases of the Skin."*)—*Antipyrin* sometimes causes a red papular eruption, not unlike measles, more rarely an urticaria.

Arsenious Acid and its preparations cause occasionally urticarial or papular eruptions, and, if long continued, a brown pigmentation of the skin.

Belladonna and *Atropin* cause a general red rash resembling that of scarlatina.

Bromides, when given for some time, cause pustular eruptions on the face, chest and back, the so-called "*Bromide Acne.*" A number of spots may coalesce to form larger patches.

Chloral Hydrate, in large doses, occasionally produces a dark red papular rash, affecting chiefly the face, neck and limbs.

Copaiba often causes a profuse rash, consisting of red, slightly raised spots, discrete or confluent, and affecting the trunk, limbs and face.

Iodides cause rashes of various kinds; the most important is the "*Iodide Acne*," a crop of pustules very like those produced by the bromides, but usually smaller and more pointed. In rare cases papules, wheals, or purpuric spots are produced.

Mercurial preparations, in rare cases, give rise to a red papular eruption or a diffuse redness with swelling.

Opium and *Morphia* sometimes cause a red papular eruption resembling measles or scarlet fever.

Quinine may produce a rash like scarlet fever, or a papular one like measles.

Silver Nitrate, after prolonged administration, causes a peculiar slaty-grey colour of the skin; this may be so dark as to be nearly black; it is of general distribution, but deeper on parts exposed to light. Once produced, the discoloration is a permanent condition.

Sulphonal has occasionally been followed by a red macular eruption.

Tuberculin, injected hypodermically, causes, with the constitutional reaction, a red eruption on the skin, either papular round the hair follicles, or red spots of varying size and distribution.

Turpentine and *Terebene* are occasionally followed by redness, papules, or even vesicles, with intense itching.

Abnormal Conditions of the Nails.

Onychauxis, or hypertrophy of the nail, is not uncommon; there is much thickening and irregularity, and the nail may form a horn-like growth several inches in length and of various shapes.

Onychomycosis is due to affection of the nail by fevers or ringworm; the affected nail is dry, lustreless, discoloured, opaque, furrowed, fissured, and raised from its bed; the parasite can usually be found by microscopic examination.

Onychia is a general term used for inflammation affecting the matrix of the nail. *Ingrowing toe-nail* is an inflammation of the skin and matrix set up by the pressure of the tissues against a sharp edge of nail; it is usually found on the great toe.

Transverse Furrows in the nails occur as the result of acute diseases, such as specific fevers; the position of the furrow affords some information as to the occurrence of recent illness, and the breadth and depth correspond roughly to its duration and severity.

Curved Nails occur often with clubbing of the fingers in patients suffering from phthisis or chronic heart disease.

CHAPTER VI.

EXAMINATION OF THE RESPIRATORY SYSTEM.

The symptoms usually complained of by patients suffering from disease of the respiratory organs are:—Pain, cough, spitting of phlegm or blood, changes in the voice, shortness of breath, loss of appetite, or more general symptoms, such as weakness, loss of flesh, &c.

Changes in the strength or quality of the voice, in association often with cough, suggest disease of the larynx; cough, expectoration, hæmoptysis, difficulty in breathing, and loss of flesh and strength, together with a varying degree of pyrexia, suggest disease of the lungs, while acute thoracic pain and shallow breathing characterise the onset of pleurisy.

The pain of pleurisy is often stabbing in character, and makes the patient afraid to breathe. Its severity is a measure to some extent of the severity of the attack of pleurisy. The pain is usually most severe before friction-sound can be heard, and often passes away as the rub becomes audible. The pain of pleurisy may be referred to the anterior

part of the abdomen, and sometimes to the groin or hip; also severe pain may be present over the tip of the shoulder and outer third of the clavicle.

It is well to remember that an attack of pneumonia is frequently ushered in, especially in children, by symptoms closely resembling those of acute gastric catarrh; thus there may be severe vomiting, belching of wind, palpitation of the heart, together with pain at mid-sternum and between the shoulders, while occasionally the patient suffers from rumbling of the bowels and diarrhœa.

ARTIFICIAL DIVISIONS OF THE CHEST.

For purposes of accurate description, it has been found convenient to mark the surface of the chest with certain imaginary vertical lines and to divide it into certain regions.

The lines are:—

1. **The median line,** drawn through the middle of the sternum.
2. **The mammary line,** drawn through the nipple.
3. **The parasternal line,** drawn midway between the preceding and the edge of the sternum.
4. **The anterior axillary line,** drawn from the anterior fold of the axilla.
5. **The axillary line,** drawn downwards from the apex of the axilla.
6. **The posterior axillary line,** drawn downwards from the posterior fold of the axilla.
7. **The scapular or dorsal line,** drawn through the angle of the scapula.

The regions are:—

Anteriorly.—1. The supra-sternal region or notch.
2. The superior sternal region or notch, separated from
3. The inferior sternal region or notch by a line joining the lower border of the third costal cartilages.
4. The supra-clavicular.
5. The clavicular, corresponding to the inner half of the clavicle.
6. The infra-clavicular, from the clavicle down to the lower border of the third rib.
7. The mammary, from the third to the sixth rib.
8. The infra-mammary, from the sixth rib to the costal margin.

Laterally.—1. The axillary, from the apex of the axilla down to the sixth rib.
2. The infra-axillary, from the sixth rib to the costal margin.

Posteriorly.—1. The supra-scapular region or fossa.
2. The supra-spinous region or fossa.
3. The infra-spinous region or fossa.
4. The interscapular, lying between the scapula and the middle line.
5. The infra-scapular.

In recording the locality of morbid phenomena, it is, however, often necessary, in order to ensure perfect accuracy of description, to state not only the region, but the particular rib or intercostal space where the physical signs are situated.

Methods.—The methods employed in making a physical examination of the lungs are :—

Inspection, including mensuration ; palpation ; percussion ; auscultation ; succussion.

INSPECTION.

Much valuable information is obtained by a careful inspection of the chest, and, whenever practicable, this should always precede other methods of investigation.

It is not unnecessary to lay stress on the importance of this mode of procedure, for many students are too apt to begin their examination with percussion and auscultation, and thereby omit to notice facts which are frequently of great aid in diagnosis.

The patient should be placed in a good light, with the surface of the chest fully exposed to view, but with the back protected by some covering. If not too ill, he should either sit or stand, in an unconstrained position, near the fire. The observer should view the chest from the front, from the back, and from either side. He should first examine the condition of the skin as well as the nature of any eruption present ; then take note of any undue visibility or distension of the superficial vessels ; and finally direct his attention to the size, shape and movements of the thorax.

Variations in the condition of the skin are described in Chapter V. ; those relating to the vessels in Chapter VII.

Size and Shape.—In surveying the chest with the eye, the following points require special consideration :—Its length or height ; the relation between the antero-posterior and transverse diameters ; the size of the costal angle (that formed by the convergence of the rib cartilages at the xiphoid cartilage) ; the direction of the ribs ; the width of the intercostal spaces ; the arching of the sternum and spine ; the height of the shoulders ; and the projection of the scapulæ.

The circumferential shape is accurately determined by means of the **cyrtometer**, an instrument readily made by uniting two long pieces of soft metal, such as lead, by a leather hinge ; the hinge is placed over the spine, the metal arms are moulded to a given circumference of the chest-wall, and are then removed without alteration in their shape by means of the hinge. When laid on a sheet of paper, an accurate tracing of the contour of the chest at the required level may be obtained. The length of the diameter of the chest is determined by calipers, and thus the cyrtometer tracing may be checked and rendered more accurate.

The Normal Chest.—In new-born children the antero-posterior and transverse diameters are nearly equal, and a tracing of the circum-

ference is almost a perfect circle. This shape is maintained till towards the end of the second year, after which it gradually passes into the elliptical shape of the adult chest, in which the transverse exceeds the antero-posterior diameter, and the front of the chest is flattened instead of being rounded, as in infancy.

As old age comes on, the chest tends to acquire the same rounded figure that it had at the beginning of life.

In a well-formed chest, the right half is slightly larger than the left half, but otherwise the two sides are perfectly symmetrical; the nipples are seated on the fourth ribs, or on the fourth interspaces, and the costal angle is nearly 90° in size. The supra- and infra-clavicular regions are nearly on the same level with the clavicles, and the outline of the ribs is usually only apparent in the lower part of the lateral regions. Deviations from a perfectly shaped chest are exceedingly common, and are compatible with the soundest health. The more marked departures from normal, either general or local, require a special description, and may be grouped as follows :—

I. Bilateral and symmetrical changes—
  A. Natural deformities—
    The alar chest.
    The flat chest.
  B. Accidental deformities—
    Transverse constriction of the chest.
    The rickety chest.
    The pigeon chest.
    Enlargement of the chest.
    Diminution of the chest.

II. Unilateral changes—
    Enlargement.
    Diminution.

III. Local changes—
    Bulging.
    Depression.

The Alar or Pterygoid Chest.—This form is characterised by an undue obliquity of the ribs, in consequence of which the thorax is elongated vertically, the shoulders droop, and the angles of the scapulæ project from the trunk like wings, hence the name "pterygoid." The antero-posterior diameter is shorter than normal, but the transverse contour of the chest is not materially altered in shape.

The Flat Chest.—In this variety there is flattening from before back, the cartilages of the true ribs are straight instead of curved, and the sternum may even be depressed below the level of the cartilages. The flat and the alar types of chest are natural deformities: they

indicate deficient capacity for lung-tissue, and have been called phthinoid chests, because persons possessing such chests frequently exhibit tubercular tendencies.

Transverse Constriction of the Chest, or Harrison's Sulcus.—This is

FIG. 71.—Chest and Abdomen of Young Child the subject of Rickets. On the right side a row of beads with a groove in front of it is seen. The projection of the sternum and costal cartilages and the enlargement of the abdomen are also shown.

a sulcus or depression which extends from the base of the xiphoid cartilage on either side, outwards and slightly downwards, and ceases at about the mid-axillary line. It is very common, is produced in childhood, and persists during later life. Frequently it accompanies

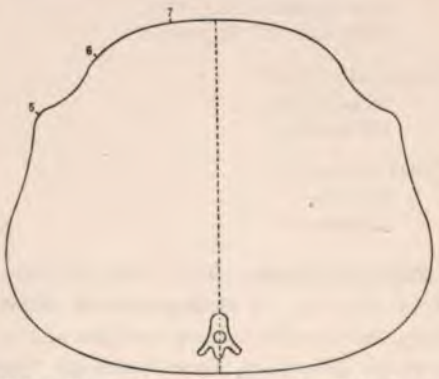


FIG. 72.—Tracing of Rickety Thorax of Boy aged 13 months. The bead (at a distance of 5 inches from the spine) and the grooves in front of and behind it are indicated.

other deformities, such as the alar, flat, rickety, or pigeon chest, but it may exist alone.

The Rickety Chest.—A typical rickety thorax is characterised—

(1.) By a row of beads at the junction of ribs with the costal

cartilages; collectively they form the "rickety rosary." These beads are most manifest about the fifth and sixth ribs; they are generally symmetrical on the two sides; they are usually distinct at the age of three months, and may increase in size up to the end of the second year, but are rarely found in children over five years of age, and no vestige of them remains in adult life.

(2.) By the presence of two grooves, one in front of the beads, the other behind them. The former is a slight groove, and is usually more easily felt than seen (see Fig. 72); the latter is a broad shallow depression, which begins outside the nipples on each side, and extends obliquely from above downwards and outwards (see Fig. 71, also Fig. 4, p. 34). Thus the contour of the rickety chest tends to resemble that of a guitar (see Fig. 73).

(3.) By increased convexity of the costal cartilages, which, with the

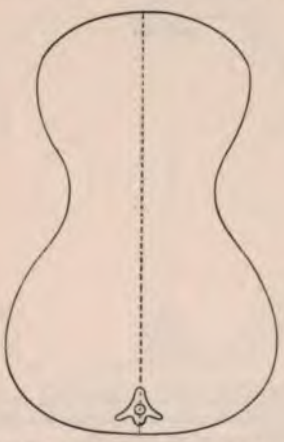


FIG. 73.—Tracing of a Rickety Thorax of Young Child, showing extreme deformity.

sternum, form a broad rounded projection. This convexity, as well as the lateral grooves, is shown by a cyrtometer tracing taken a little below the level of the nipples.

(4.) By the presence of Harrison's sulcus.

The Pigeon Chest.—The essential feature of the pigeon chest is that the outline of a horizontal section approximates to the triangle, the true ribs being straightened in front of their angles and the sternum carried forward. This type of thorax may be found without any beads or other signs of rickets. It occurs most commonly in children over two years of age who have suffered from some chronic respiratory trouble which interferes with the entrance of air; and, unlike the rickety, the pigeon chest often persists in adult life.

Bilateral Enlargement of the Chest.—This is nearly always the

result of emphysema of the lungs. It may be called the inspiratory chest, but in a well-marked case the enlargement is much greater than can be produced with healthy lungs by the deepest inspiration. The antero-posterior diameter is increased, and may even exceed the transverse diameter. The sternum is projected forwards and the spine is arched backwards; sometimes the former predominates, sometimes the latter. The projection backwards is constituted by the spine and the angles of the ribs; immediately in front of it, and involving the scapular and infra-scapular regions, the chest-wall is flattened, or even slightly depressed. Hence a horizontal outline is not truly circular, and the term "barrel-shaped" scarcely gives an accurate idea of the emphysematous thorax.

Sometimes the enlargement is limited to the upper part, but more commonly it involves the whole length of the chest; in the latter case

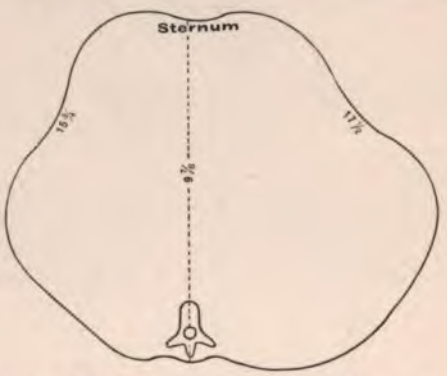


FIG. 74.—Excessive Arching of Right Ribs in a case of Syringo-myelia. The increase in size of the right side of the chest has slowly developed during the last fifteen years, and has been accompanied by overgrowth of the bones of the right limbs.

the costal angle and the lower intercostal spaces are much wider than normal, and the cartilages of the false ribs are everted.

Paralysis of the diaphragm is another occasional cause of general enlargement of the chest.

A chest closely resembling that of emphysema sometimes occurs as a consequence of habitual stooping; also in association with marked stooping and permanent alteration in the shape of the spine—spondylitis deformans of the vertebræ and rib articulations due to ankylosis.

Lengthening of the antero-posterior diameter of the chest may also result from caries of the vertebræ.

Bilateral Diminution, in which the capacity of the thorax is less than that of a healthy one in a condition of deepest expiration, may result from (1) phthisis or (2) from paralysis of the intercostal muscles.

Unilateral Enlargement.—This is most commonly caused by the presence of serum or pus in the pleural cavity, but is also met with as a result of pneumothorax, of extensive hæmothorax, of a tumour affecting the greater part of one lung, or of compensatory hypertrophy in consequence of chronic disease of the other lung. Enlargement of the affected side is indicated by: elevation of the shoulder and ribs; widening of the intercostal spaces and of one half of the costal angle, and deviation of the spine towards the opposite side.

The side looks rounder than its fellow; there is a tendency, *e.g.*, in pleural effusion, for the affected side to assume the semicircular form, and the vertebro-mammary diameter is increased in length. A peripheral measurement, however, may not be greater than that of the healthy side, which is also enlarged to a slight degree, for it must be

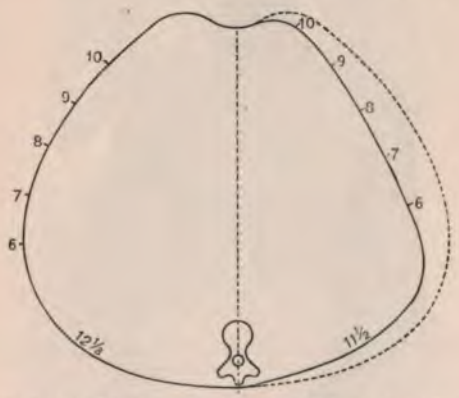


FIG. 75.—Tracing of Thorax in a Case of Fibroid Phthisis affecting the right lung. The figures indicate the distance in inches from the spine; the dotted line indicates the probable normal shape of the right side. The greatest flattening was in the axilla. The cardiac impulse in this case was in the fourth right intercostal space a little outside the nipple line.

remembered that any centrifugal pressure on one side will affect the chest as a whole, there being no unyielding partition between the two halves. The cyrtometer is of value in showing a *localised* rather than a general effusion.

Unilateral Diminution.—The common causes of retraction of one half of the chest are—(1) pleurisy, the fluid having been absorbed and the lung not in a condition to expand; and (2) fibroid phthisis. Rarely an infiltrating cancer of the lung leads to retraction of the chest-wall. Occasionally, and especially in children, a rapid shrinking of one side follows collapse of the lung due to obstruction of the main bronchus.

On viewing such a chest from before or behind, the affected side looks flat, the shoulder and ribs are depressed, and the nipple is on a

lower level than its fellow; the intercostal spaces are narrowed, and the inferior angle of the scapula is lower and nearer to the spine than that on the healthy side. The spine too is curved towards the healthy side.

The distortion due to pleurisy with retraction is usually more marked than that produced by cirrhosis or collapse of lung. An apparent unilateral diminution may be produced by lateral curvature of the spine. In such a case, however, a flattening in front of the chest is

FIG. 76.—Retraction of Left Side of Chest following an Empyema which perforated the chest-wall below left scapula fifteen years ago. Now the left side is dull from apex to base; the extent of dulness to the right is indicated by the dotted line.

compensated for by a protuberance of the chest-wall behind or *vice versâ*.

Local Bulging of the chest-wall may be the result of one of the following conditions:—Tumours, &c., of chest-wall; a circumscribed pleural effusion; a pointing empyema; pericardial effusion; hypertrophy and dilatation of the heart; an aneurysm; a hernia of the lung; very rarely a large phthisical cavity or an intrathoracic growth. A bulging of the lower part of the chest-wall on the right side may be caused by an enlarged liver; on the left side by an enlarged spleen.

Local Shrinking.—The most familiar example is the depression of the supra- or infra-clavicular region which follows shrinking of the

FIG. 77.—Shows scar of old abscess below left scapula, and ink line drawn over spinal column to show the curvature.

FIG. 78.—Tracing of Chest-Wall of man photographed in Figs. 76 and 77.

apex of the lung in phthisis; and in inspecting the chest-wall, particular attention should always be directed to these regions, a slight flattening

below the clavicle or a slight depression above it being significant signs of early phthisis. Rarely, flattening may be simulated by atrophy or congenital absence of part of the pectoral muscles.

A cup-shaped depression, involving the lower part of the sternum and attached cartilages, and varying in height and depth, is of frequent occurrence. It may follow some chronic form of obstruction to the

FIG. 79.—Photograph of Boy the subject of Phthisis at the right apex, to show the hollows above and below the clavicle, the dropping of the right shoulder and the slight lowering of the right nipple as compared with the left. The shaded area indicates the extent of impaired resonance, the darkest shading indicating where impairment was greatest.

entrance of air into the chest, such as adenoids, enlarged tonsils, hooping-cough, pericardial adhesion, or, according to Gee, a unilateral pleurisy. Sometimes it is produced by pressure, as in shoemakers. In many cases it is difficult to find a satisfactory explanation for this deformity.

Movement.—Changes in the respiratory movements of the chest-wall may be considered under the following headings:—

Increase of respiratory movement.
Decrease of respiratory movement.
Respiratory retraction of chest-wall.
Alterations in rhythm and rate.

Increase of Respiratory Movement affecting the whole chest is observed along with accelerated breathing in the pyrexial state, also in hysteria. Unilateral or local increase occurs when the opposite lung is incapacitated by reason of changes in its tissue, fluid in its pleural cavity, or obstruction of its bronchus.

The movement of the diseased side being usually less than normal, renders the asymmetry of the two sides still more striking.

In phthisis, when both apices are diseased, the respiratory movement of the lower part of the chest is exaggerated. Conversely, when air does not freely enter the lower parts of the lungs, the upper part of the chest shows increased movement.

Decrease of Respiratory Movement occurs—

1. When there is any hindrance to the entry of air into the respiratory passages. This may be caused by laryngeal disease; by compression of the trachea or bronchi; by imperfect expansion, collapse, or consolidation of the pulmonary vesicles, as from phthisis, pleural effusion, or any painful affection of the chest-wall.

2. When the air-cells are unduly and permanently distended, rendering them incapable of much further enlargement by an inspiratory effort. This condition characterises emphysema, in which, as Jenner puts it, "the patient tries to take in his breath at the top of his breath."

3. When the muscles of respiration are weakened or paralysed. In hemiplegia feebleness of chest movement on the affected side may not be visible during easy breathing, but it usually becomes apparent when the patient draws a deep breath.

It is to be observed that, associated with lessened extent of movement, there is usually delay in time. On watching, for example, the infra-clavicular regions in a case of phthisis at one apex, it may frequently be noticed that the diseased side not only expands to a less degree than the healthy side, but lags behind it, and takes a longer time to complete its excursion. Indeed, the alteration in relation to time may sometimes catch the eye before that in relation to space.

In paralysis of the intercostal muscles, respiration is carried on by the diaphragm, and by the extraneous muscles which elevate without expanding the upper part of the chest. In paralysis of the diaphragm the epigastrium is hollowed, and is drawn in instead of being protruded during each inspiration, while during expiration it may be slightly

protruded instead of falling in, as in normal breathing. There is also overaction of the lower intercostals, so that in quite a remarkable way the margin of the thorax moves with respiration far more than in ordinary breathing. This may be well seen in some cases of alcoholic and diphtheritic paralysis.

Respiratory Retraction of the Chest-Wall.—Severe obstruction to the entrance of air into the respiratory passages may lead to more than mere abolition of respiratory movement; there may be movement in a direction opposite to normal—that is, certain parts of the chest-wall become drawn in with each inspiration. Thus, when the apex of the lung is solidified and excavated, the supra- and infra-clavicular regions may sink in during inspiration; and in severe laryngeal obstruction, recession of the front and sides of the thorax below the level of the nipples is usually conspicuous (see Inspiratory Dyspnœa). Normally,

FIG. 80.—From a Fatal Case of Alcoholic Paralysis a few hours before death, showing the retraction of the epigastrium which occurs in paralysis of the diaphragm. (*Ross.*)

there is indrawing of the intercostal spaces during inspiration. In pleurisy this feature is often absent; the part remains immobile, if not actually bulged.

Rhythm and Rate.—The average rate of breathing in the adult male is from sixteen to twenty per minute. It is somewhat quicker in females and in children. The new-born infant takes about forty-four respirations per minute, a child three years old about twenty-five per minute, one of fifteen about twenty.

Both rhythm and rate are much more easily disturbed in the child than in the adult, and hence, relatively, are of less value. They are apt to be disturbed if the patient's attention be directed to the respiratory act, hence it is better to determine the frequency of respiration by inspection than by palpation; and it is convenient to watch the movements of the chest after counting the pulse, and while the finger still remains upon it.

Dyspnœa is a term somewhat loosely applied to all instances of difficult breathing, in which, as a rule, there is increased frequency of respiration. **Orthopnœa** signifies very great difficulty in breathing, causing the patient to assume a sitting or standing posture. It is seen during the paroxysm of asthma, and is often present in the severer forms of heart disease.

In investigating a case of disordered breathing, it is necessary to observe the frequency, the depth or shallowness, and any peculiarity in the rhythm of the respiratory act.

Diminished Rapidity of Breathing may be met with in all severe affections of the brain or its membranes, as tumours, extensive hæmorrhage, and any variety of meningitis. As a rule, stupor or coma is present, and the respirations, although slower, are deeper than normal. Frequently, in such cases, the breathing tends to assume the Cheyne-Stokes type (see p. 141). In diabetic coma respiration is often slow, deep and sighing in character. Where slowness of breathing is accompanied by obvious effort or discomfort to the patient, the term dyspnœa may correctly be given to it. This laboured respiration is especially marked in stenosis of the larynx or trachea, from tumour, inflammation, compression, or other cause.

Increased Rapidity of Breathing occurs—

1. In pyrexial conditions. The degree of acceleration varies with the nature of the febrile disease, and to some extent in different individuals. Nervous persons and children breathe more quickly than others suffering from the same degree of pyrexia. Any great increase in the frequency of respiration during the course of a fever should lead to a careful examination of the lungs and heart.

2. Whenever breathing is attended by pain. This is the case in disease of the pleura; in inflammation of the diaphragm or of the peritoneum, especially that portion covering the diaphragm; and in painful affections of the thoracic walls, such as pleurodynia, or injury to the ribs. In this class of cases the chest movement, although accelerated, is shallower than normal.

3. In diseases of the bronchial tubes, whether obstructed by secretion as in bronchitis, or narrowed as in asthma.

4. In all conditions which either diminish the breathing surface of the lungs or hinder their proper expansion. This includes all diseases of the lungs, pleuritic effusion, pneumothorax, mediastinal tumours, abdominal affections which raise or hinder the descent of the diaphragm, deformities of the thorax which lessen its capacity, paralysis or spasm of the inspiratory muscles, as in tetanus or epilepsy.

5. In diseases of the heart or great vessels which lead to congestion

of the pulmonary circulation. Of these, mitral disease and a clot in the pulmonary artery may be specially mentioned.

6. In diseases of the nervous system. The slow laboured breathing of many brain affections has been already mentioned. In other cases great rapidity of breathing may be observed, as in the terminal period of many forms of meningitis.

In hysteria, acceleration without any real difficulty in breathing may be observed; but when there is laryngeal spasm, violent paroxysms of dyspnœa occur and respiratory distress is often extreme.

7. In abnormal conditions of the blood. Hurried respiration occurs in uræmia, and recurrent attacks of dyspnœa in cases of Bright's disease suggest the onset of uræmic coma or convulsions. Accelerated breathing occurs sometimes in diabetic coma. Quickened breathing after slight exertion, often called shortness of breath, is a marked feature of anæmia.

Inspiratory Dyspnœa signifies that there is a hindrance to the free ingress of air. It is a striking phenomenon in obstructive laryngeal disease, as from inflammation, diphtheria, or the paroxysm of whooping-cough, or of laryngismus stridulus, and occurs perhaps in its purest form in paralysis of the posterior crico-arytænoid muscles (the dilators of the glottis). Sometimes, too, it is present in hydrothorax and in acute œdema of the lungs. Drs. Barlow and Lees have also described inspiratory dyspnœa with stridor in cases which they suggest are caused by driving in of the arytæno-epiglottidian folds, owing to relaxation of tissue about these folds.

The chief sign of this condition is recession of the more yielding portions of the thoracic parietes. This is best seen in the pliable chests of young children, and when the natural pliability is increased, as by rickets, a very slight obstacle to breathing, such as that produced by a mild attack of bronchitis, is often sufficient to induce recession of the chest-wall.

With each inspiration the lower end of the sternum and the epigastrium, together with the lower lateral regions, are sucked in, and when the obstruction is in the larynx or upper portion of the trachea, the supra-sternal notch and the supra-clavicular regions are also depressed. At the same time the sternum is pushed forward, and, by watching such a case, the mode of production of the pigeon chest and of Harrison's sulcus is easily appreciated. With respect to rhythm, inspiration begins abruptly and is prolonged, while expiration is shorter than natural, but the pause relatively to the respiratory act is longer than natural.

Expiratory Dyspnœa signifies that there is a hindrance to the free egress of air. This may be due either to a diminution in the ex-

piratory power of the lungs or to a lesion obstructing in a certain way the upper air passages. The former condition holds in emphysema and asthma; the latter is illustrated by a movable tumour situated immediately below the glottis, which is forced against the glottis by the expiratory current, but pushed away from it by the inspiratory current of air. In emphysema, especially when complicated by bronchitis, the diaphragm descends powerfully during inspiration, but thoracic movement is almost limited to elevation, there being little or no expansion, while expiration is slow, laboured, and prolonged.

Cheyne-Stokes Breathing.—This designation is given to a peculiar alteration of rhythm, in which respiration occurs in repeating cycles; each cycle is composed of an ascending and a descending phase, and is succeeded by a period of complete cessation of respiration. The ascending phase begins with the shallowest possible inspiration, this is succeeded by a number of inspirations of gradually increasing depth till the acme is reached, when the descending phase commences, which

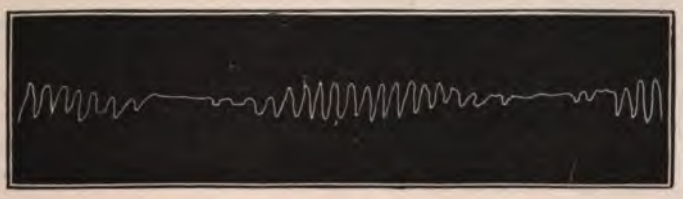


FIG. 81.—Tracing from a Case of Cheyne-Stokes Breathing. (*Gibson.*)

comprises a number of respirations of gradually decreasing depth till breathing stops. The pause may last for from five to forty seconds, the cycle from fifteen to seventy-five seconds. During the pause the pupils often contract, the pulse becomes slower, and twitching movements of the limbs may be observed. This phenomenon is met with in meningitis, apoplexy, heart disease, uræmia, and in some acute diseases, as typhoid fever. Occasionally it passes away and the patient recovers for a time, or even completely, but, as a rule, the prognosis is unfavourable, and indeed the symptom usually appears only a short time before death.

PALPATION.

By palpation, or the application of the hand to the surface of the chest, the results obtained by inspection with regard to the shape, size and movements of the chest may be checked and amplified, and the following points may also be investigated:—The nature and situation of any sensory disturbance; the presence or absence of

fremitus, whether vocal, tussive, rhonchal, or friction; of fluctuation and of pulsation.

Movements.—The palms of the hands or fingers must be applied evenly and on symmetrical portions of the chest-wall; in this way the anterior, posterior and lateral movements may be examined. The movements of the upper part of the chest are best felt (1) by placing the hands on the surface below the clavicles, so that they diverge from one another with the tips of the fingers touching the outer part of the clavicles; (2) by standing behind the patient and placing the hands over the shoulders, so that the fingers lie on the infra-clavicular regions. To investigate the posterior movements the hands should be laid over the interscapular and scapular regions, while the lateral movements may be felt by grasping the sides of the chest from before or behind. The strength of the diaphragm, as well as the relative strength of its two halves, may be tested by applying the hands to the abdomen, so that the finger-tips cross the epigastrium.

The conditions in which deficient or excessive movement occurs have been already enumerated under "Inspection," but attention may here be drawn to the importance of distinguishing between elevation and expansion of the thoracic parietes. To the eye the thorax of severe emphysema may appear to move sufficiently well, but to the hand it is evident that, while it is raised as a whole, there is little or no filling out of its walls—that is, no real expansion.

Sensory Disturbance.—Indications of disease affecting the parietes or contents of the thorax are frequently afforded by touching or pressing the surface. Thus it may be found on touching or pinching the skin that certain parts of the surface are less sensitive or more sensitive than normal; for example, diminution or loss of cutaneous sensibility over one half of the chest points to disease in the central nervous system—a band of increased sensibility or hyperæsthesia extending across and around the chest suggests irritation of the posterior roots of some of the spinal nerves.

Localised spots of tenderness may be due to obvious lesions of the skin or bone, or to an affection of muscular tissue. Tender spots are present along the course of one or other of the intercostal nerves in neuralgia, as, for example, that associated with shingles; they occur also in hysteria. A more localised tenderness, situated in an intercostal space, especially in the axillary region, may be observed in early pleurisy; sometimes, too, during the stage of effusion, particularly if the fluid be purulent. In examining the chests of children suffering from pneumonia, it often happens that they shrink or cry during percussion or auscultation of the affected side; the same thing may be observed to a less degree at the apex in phthisis.

Fremitus.—Vocal Fremitus in Health.—The vibrations of the vocal cords produced by the voice in speaking, singing, or screaming, are communicated to the chest-wall, where they are distinctly felt by the hand in the vast majority of healthy persons. The intensity of vocal fremitus depends on the following conditions:—

(1.) On the strength and pitch of the voice, loud and low-pitched voices yielding more marked fremitus than weak and high-pitched ones. Hence this sign is comparatively of less value in women and children than in men.

(2.) On the size of the bronchus and its position relatively to the chest-wall. Vocal fremitus is therefore usually more intense on the right than the left side, the right bronchus being wider and nearer to the back than the left one. The difference between the two sides is best marked below the clavicle, below the scapula, and between the scapula and the spine, and it is in these regions that deviations from normal are best appreciated.

(3.) On the distance of the examined spot from the larynx; fremitus being strongest over the larynx, and more marked over the upper than the lower regions of the thorax.

(4.) On the thickness of the chest-walls; thus the vocal thrill is less perceptible over fat, muscular, or œdematous chests than over thin ones.

Vocal Fremitus in Disease.—In examining the vocal thrill, all patients should be asked to repeat the same sound, the words "ninety-nine," pronounced in as deep a tone as possible, being convenient ones for the purpose; and in comparing the two halves of the chest, care should be taken to place the hands or the tips of the fingers on symmetrical spots. Under certain morbid conditions the vocal fremitus may be diminished, abolished, or increased.

Vocal Fremitus is Diminished or Abolished—

(1.) When there is an effusion of liquid or gas in the pleural cavity.

In pleurisy the presence of effusion may be determined and its extent mapped out by the alteration in vocal fremitus; above the effusion the fremitus is normal or increased, the increase being often marked in the infra-clavicular region; more fluid is required to annul fremitus over the right than over the left infra-scapular region. The return of fremitus to a part is often the first indication that absorption of pleuritic exudation has commenced.

(2.) In very dense consolidation of lung tissue, whether the result of inflammation or of new growth, unless the solid mass be intimately connected with a large open bronchus.

(3.) Over lung tissue, either healthy or diseased, when its bronchial tubes are narrowed by secretion, external pressure, or other causes.

Thus the vocal thrill is sometimes feeble in chronic bronchitis and in asthma; occasionally it is diminished, or even completely absent, over a pneumonic lobe or a phthisical apex.

Vocal Fremitus is Increased—

(1.) In all forms of lung consolidation, provided the solidification is not too dense and is traversed by air tubes, the large ones at least being unobstructed.

(2.) Over pulmonary cavities which are near the surface, have thick walls, and communicate with open bronchi.

The question of vocal fremitus is largely one of comparison between the two sides of the chest, and it is a valuable aid to diagnosis between pleuritic effusion and pneumonia of the inferior lobe of the lung. It cannot, however, for reasons already indicated, be relied on alone. In a child the cry is a valuable means for testing fremitus, but it must be admitted that the thrill produced by voice or cry is often so misleading that the exploring syringe has to be used before a certain diagnosis can be made. The vibration produced by coughing—**tussive fremitus**—is less marked than that of the voice.

Rhonchal or Bronchial Fremitus.—Narrowing of the bronchial tubes produces sounds known as rhonchi (see p. 161). Their vibrations are often readily felt, and are particularly common in children.

Friction Fremitus.—The vibration produced by the rubbing together of inflamed pleural surfaces is sometimes transmitted to the chest-wall, where it may be felt as a vibratile rubbing or grating sensation. It is commoner during the later than the earlier periods of pleurisy.

Fluctuation.—The sensation of ordinary fluctuation may occasionally be detected in cases of empyema which lead to bulging of the intercostal spaces. Fluctuation by percussion of the surface may also in rare cases be of diagnostic aid, as when a large pleural effusion is found in association with an intrathoracic tumour.

Pulsation.—Rarely in cases of left empyema pulsation of cardiac rhythm may be perceptible, and is usually situated somewhere between the left clavicle and the sixth rib; the heart is always much displaced to the right. Aneurysm may be simulated by a pulsating empyema; very rarely it may be necessary and justifiable to use a fine hypodermic syringe in order to make a diagnosis.

PERCUSSION.

Percussion is the art of striking the external surface of the body—chiefly of the chest and abdomen—in order to ascertain the physical condition of the underlying parts. To this end the nature of the

sound emitted by the percussed part, and the degree of resistance offered by it and felt by the observer, must both be carefully studied.

There are two methods of percussion—the immediate and the mediate.

Immediate Percussion is performed by striking the chest with the palmar surface of the fingers, or with the tips of two or three brought together in the form of a hammer. It is sometimes used to obtain a rough preliminary notion of the limits of the intrathoracic organs, or of the presence and extent of pathological changes. Thus in effusion into one pleural cavity, the broad contrast between the sounds on the two sides may be easily and quickly demonstrated. Its main use, however, is in the percussion of certain bony prominences, as the clavicle or spine of the scapula, the bone being lightly tapped with one finger.

Mediate Percussion may be performed either (1) by means of a small hammer with its striking end tipped with india-rubber, and a pleximeter consisting either of a thin piece of ivory or of one of the fingers; or (2) by the fingers only. The latter is by far the most convenient and precise method, for not only does it satisfactorily discriminate between the finer gradations of sound, but also supplies information by the sense of touch with regard to degrees of resistance and elasticity. In its employment one finger of the left hand —usually the first or second, or sometimes, as in percussing above the clavicles, the little finger—must be placed with the palmar surface of the last two phalanges accurately and firmly applied to the part. This pleximeter finger is then struck with the semiflexed first or second finger of the right hand. Observe also (1.) that when comparing the sides of the chest or two parts of the same side, the pleximeter finger must be applied precisely in the same manner as regards pressure and direction, and over similar structures; thus if placed vertically over one spot, it must be placed vertically over the compared spot; rib must be compared with rib, intercostal space with intercostal space. (2.) The percussion stroke must spring from the wrist only; thus the force of the blow may be regulated with nicety, and so made equal in any two compared spots. (3.) The blow should be delivered quite vertical to the surface percussed; as a rule, it should be light, for if too strong the vibration of the neighbouring parts may confuse the true sound. This is especially the case in children, owing to their yielding thoracic walls.

Theory of Percussion.—All sounds are divided into noises and musical sounds or tones. The vibrations that constitute the latter are repeated at regular intervals, are periodical, each has the same wave-length; those of the former succeed one another irregularly, without

periodicity, the consecutive vibrations are unlike. Between a pure musical sound and a harsh noise there is, however, no abrupt separation; many intermediate sounds bridge over the gap between them. Thus the sound emitted by the healthy chest is neither a noise nor a perfect musical note; it possesses tone, but this is made up of a series of tones which do not completely harmonise; the musical quality is therefore impaired. The various percussion sounds, normal and abnormal, might also be divided into tones and noises: the former possess the common properties of loudness, duration, pitch and tone; in the latter, tone is absent and pitch for the most part is indistinguishable. But in clinical reports we are in the habit of describing percussion sounds not as "tones" or "noises," but as possessing varying degrees of **resonance** or of **dulness**. For resonance is the production of tone by rhythmical reflection, and since, as Dr. Gee points out, "the only tones which percussion knows (those of bone and cartilage excepted) are produced by resonance" (that is, by the rhythmical vibrations in the cavities formed by the lungs or by the alimentary canal), it has come about that "the words tone and resonance as applied to percussion sounds mean the same thing."

The important points to be observed in percussion are—

1. The degree of clearness of the tone or resonance.

2. Next to tone, pitch is the most important quality; and of the two remaining qualities duration is of more practical value than loudness. Pitch and duration bear an inverse relation to one another; thus the shorter the duration the higher the pitch.

3. The resistance felt.

To become familiar with simple standards, and with the chief variations in the sounds and the tactile sensations of percussion, the student may profitably go through the following **exercise**, practising on himself or on a fellow-student:—

(1.) Let him percuss the fleshy part of his own thigh and note its resistance and tonelessness; this is the best example of dulness.

(2.) Percuss his stomach, this gives *clear*, as distinct from muffled, resonance. Contrast the stomach with the colon as regards pitch.

(3.) Percuss the right front, and observe the normal muffled quality of the thoracic resonance. In percussing downwards over the mammary region, observe the rise in pitch and the shortening of the duration of the note, also the increase in resistance on coming to the liver.

(4.) Percuss out the cardiac dulness, carefully noting how much of the third and fourth spaces are impaired, reckoning from the left margin of the sternum outwards to the left.

(5.) Fillip or percuss over the pomum Adami with the mouth open,

and note the pure tubular sound; contrast its pitch and duration with those of the right mammary region.

(6.) Percuss a fellow-student's back close to the spine, then pass outwards, noting the increase of resistance when over the muscles.

(7.) Percuss the clavicle or the back of the second phalanx; observe that the pitch of this osteal note is higher than the note over the trachea, and that the latter is higher than that over the thorax.

(8.) Percuss a lung in the post-mortem room, and observe the clear tubular character of the note.

Limits of the Lungs.—Each lung, somewhat conical in shape, has three surfaces, an outer convex, an inner concave, and a lower or basal

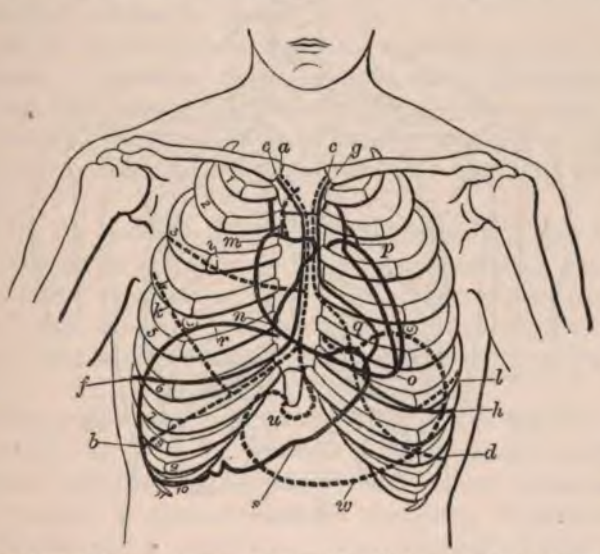


FIG. 82.—Relations of Thoracic Organs. (*Weil and Luschka.*) *a*, *b*, edge of right, *c*, *d*, edge of left pleural sac; *e*, *f*, margin of right, *g*, *h*, margin of left lung; *i*, upper, and *k*, lower fissure of right lung; *l*, fissure of left lung; *m*, *n*, right; *n*, *o*, lower; *p*, *o*, left margin of heart.

concave surface which rests upon the diaphragm; and three borders, namely, a thin anterior or median, a thick posterior, and an inferior border, consisting of an outer convex part and a smaller inner concave part.

In Health the apices rise anteriorly from 1¼ to 2 inches above the clavicles, but posteriorly they do not project above the limits of the bony thorax. Starting behind, the upper limit of each lung is marked by a line, curved with its convexity downwards from the spine of the seventh cervical vertebra to the outer edge of the trapezius, thence with a slight inward curve to the outer edge of the sterno-mastoid, and then downwards to end at the sterno-clavicular articulation. The anterior

margins lying deeply behind the sterno-clavicular articulations, descend to meet at the level of the second costal cartilages, they run parallel as far as the fourth cartilages, the right lung slightly overstepping the middle line, the left keeping near the left edge of the sternum; at the fourth cartilage the left curves outward across the fourth space to the fifth cartilage, thence it inclines towards the sternum, nearly reaching the inner third of the sixth cartilage, where it joins the lower edge to form a tongue-shaped process of lung. The position of the lower margin of the right lung during quiet respiration is as follows:—It reaches the sixth rib near the sternum, the seventh rib in the mammary line, the

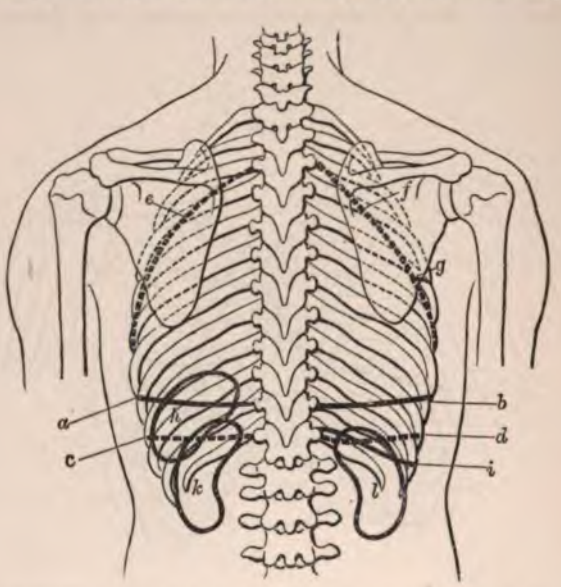


FIG. 83—Limits of Lower Margins of Lungs and of Pleural Sacs behind. (*Weil and Luschka.*)
a, *b*, lower margins of lungs; *c*, *d*, lower edges of pleural sacs; *e*, *f*, *g*, fissures between lobes; *i*, lower edge of liver.

seventh space in the axillary line, the ninth rib in the scapular line, and is opposite the tenth or eleventh dorsal spine, near the vertebral column. The left lung reaches the eighth rib in the axillary line, and the same places behind as the right lung.

The fissure between the upper and lower lobes begins opposite the third dorsal vertebra or the spine of the scapula; it passes downwards and forwards over the scapula to reach the sixth space, and terminates usually at the anterior end of the seventh rib. On the right side a second or upper fissure leaves the lower one at the posterior axillary edge about $2\frac{1}{4}$ inches above the angle of the scapula, and passes horizontally forwards at the level of the third space to reach the

anterior edge of the lung near the sternal end of the fourth cartilage. Behind, then, we percuss over the upper lobes down to the third ribs, and over the lower lobes below them. In front on the left side we percuss over the upper lobe only, on the right side mainly over the upper and middle lobes, and in the right axilla over the three lobes.

In Disease.—A difference in the height of the two lungs is so exceptional in health that it almost always suggests disease; shrinking of an apex is an early physical sign of phthisis; undue size points to emphysema, when the apices may reach as high as $2\frac{1}{2}$ inches above the clavicles. The position of the lower and other boundaries of the lungs is also similarly affected by disease.

The site of the middle lobe shows the importance, when there is chest disease, of percussing the right axilla, for in pneumonia it may be the only lobe affected. A knowledge, too, of the position of the fissure between the upper and lower lobes enables one to judge how far pneumonic consolidation is limited to one of them; but it is to be observed:—

(1.) That the anatomical limits of the affected lobe may be overstepped without its fellow being necessarily involved (a lobe in the condition of red hepatisation being larger than a healthy one). (2.) That the upper portion of the lower, and the lower portion of the upper lobe, may be picked out and glued together by a typical croupous pneumonia.

Respiratory Movement.—In quiet breathing the position of the margins varies but little, but between a full inspiration and a full expiration the difference is considerable; in the lateral regions as much as 3 to 4 inches. A diminution of the respiratory excursion to percussion is observed in emphysema, in commencing pleurisy, and in adhesion of the pleura.

The Normal Pulmonary Sound, called "**sub-tympanitic,**" is *sui generis*, and is only to be learnt by experience. It is short, rather low in pitch, and its tone is muffled.

Its nature is complex, but it is mainly made up of the vibrations of the thoracic parietes and those of the air columns in the lungs. Dr. Bristowe considers that the sound is almost entirely due to the former; other authors believe the latter to be the essential cause, and some refer the sound yielded by percussion to vibrations produced in the pulmonary vesicles and the smallest bronchioles, while others, as Dr. Gee, refer it to the vibrations of the air in the larger bronchial tubes, the sound being muffled by the intervening lung substance.

The quality varies considerably in different individuals, and to a slight degree in different parts of the chest. Hence the importance of

comparing the percussion sounds emitted by corresponding points of the two sides of the same chest.

The note is clearer in thin than in fat people; it is prolonged, low in pitch, and its resonance increased over the elastic chest-walls of the child, whereas over the rigid chest of old age the resonance is diminished and the pitch is raised. It is also less muffled—that is, clearer and purer—in the child than in the adult.

Regional Percussion.—The sound is clearer in quiet than in forced breathing. It is clearer in front and at the sides than behind, clearer above than below the clavicles, clearer below than over the scapulæ. It is shorter and higher pitched over the clavicles, ribs, sternum and scapular spines than over the soft parts. There is also a slight difference between the two apices, percussion close to the clavicle giving a less resonant note on the right than on the left side. The reverse obtains, however, below the second rib. Certain viscera encroaching on the lower pulmonary regions modify or lessen the extent of the normal sound. Thus on the right side the liver dulness is detected in the fifth space in front, the seventh in the axilla, and the eleventh posteriorly, and with hard percussion even a space higher, the lung resonance being muffled and higher in pitch in the fourth, sixth, and tenth spaces. On the left front the cardiac dulness is obtained in the fourth and fifth spaces, and it modifies the pulmonary sound to deep percussion in the third space. In the sixth space we get the tympanitic stomach note, which, about the margin of the thorax, passes into that of the colon. In the left lateral region the splenic dulness is encountered between the eighth and eleventh ribs.

It is important to observe that a normal pulmonary sound does not necessarily exclude disease of the respiratory organs; thus it may accompany a severe bronchitis, a dry pleurisy, a circumscribed lesion of the lung deeply seated, or a disseminated tuberculosis in the early stage.

Increased Resonance.—**1. Hyper-Resonance or Tympanitic Resonance.**—The loudness and duration of the typical pulmonary sound is increased, and its pitch is usually lowered, while the degree of clearness varies. In emphysema the sound tends to become clearer, but it is rare to get Skodaic resonance. Its extent is often much increased; thus resonance may be obtained down to the costal margin in front, and to the twelfth rib behind. A local emphysema around tubercular nodules at the apex may completely mask any dulness which would otherwise result from their presence.

In pneumothorax the resonance becomes tympanitic, but in cases of extreme distension it is muffled or almost abolished; when liquid is present as well as air, the position of the dulness will vary to some extent with that of the body. Occasionally the sound is amphoric.

2. **Tracheal Resonance**, or the so-called *tympanitisch* of Skoda, hence sometimes called "**Skodaic resonance**." The note is short, the pitch raised, and the tone clear. It is heard—

(1.) Over **relaxed lung.** (*a.*) In the neighbourhood of extensive infiltration of the lung, or of effusion into the pleura or pericardium. Thus in pneumonia of the lower lobe, or when there is effusion into the pleural cavity, a clear and sometimes quite a tubular or even amphoric sound may be obtained over the apex on the same side. (*b.*) Over a portion of lung which is only partially infiltrated, solid or liquid being mingled with air-containing tissue. Examples: the first and third stages of a croupous pneumonia, especially in children; œdema of the lung; catarrhal pneumonia.

(2.) Over **air spaces.** (*a.*) Smooth-walled cavities, which are either close to the surface or are separated from it by dense solid tissue. Examples: bronchiectasis; tuberculosis. If the cavity communicates freely with a bronchus, the pitch rises when the mouth is open, falls when it is shut. (*b.*) When the bronchus or natural air spaces are directly connected with the surface by solid masses. Examples: pulmonary tumours; pneumonia of the upper lobes.

(3.) Sometimes when the **diaphragm is pushed up** in consequence of abdominal distension, the increased clearness of the pulmonary resonance being probably due to relaxation of the lung-tissue. In other cases, however, the pulmonary resonance becomes impaired. This may be sometimes observed on the left front when the heart is enlarged and also raised by ascites. Here probably a portion of the left lung has, through collapse, become completely airless.

Diminished Resonance.—The note is shorter, more muffled, and its pitch is usually raised. The various degrees in which the normal pulmonary resonance may be impaired or muffled are often spoken of as (1) slight impairment or slight dulness; (2) moderate impairment or moderate dulness; (3) absolute dulness.

Diminished resonance is met with under the following conditions, passing from without inwards:—

1. In thickening of the superficial tissues, as from great muscular development, an excess of fat, or œdematous swelling.

2. When the ribs are strongly arched, as in kyphosis. A familiar instance is offered by percussing the back of a well-marked emphysematous thorax; the projecting angles of the ribs often give a moderately dull note, while the shallow depression over the infraspinous region furnishes increased or even tympanitic resonance.

3. When there are fluids or solid masses between the lung and the chest-wall.

(*a.*) In hydrothorax or dropsy of the pleura the effusion is usually

bilateral; hence there is dulness at both bases, which is commonly limited to the posterior aspect of the chest, but is not always at the same level on the two sides.

(*b.*) In pleuritic effusion the dulness is usually unilateral. It may be limited to the lowest part of the chest behind, or extend over the whole of one side from apex to base. When a moderate quantity of fluid is present, the dulness is always higher posteriorly than anteriorly, and, if the patient has not been confined to bed, there may be impaired

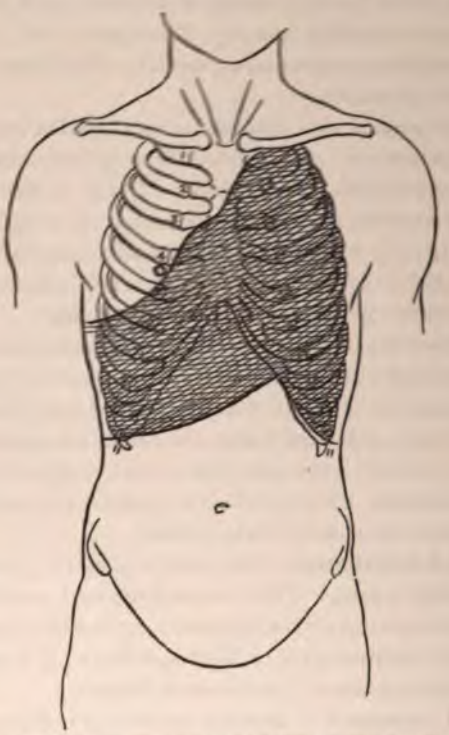


FIG. 84.—Displacement of Mediastinum, Heart, and Liver in a case of Pleuritic Effusion on the left side. The shading indicates the extent of dulness to percussion.

resonance over the greater part of the back, while the lateral and front parts of the chest are quite resonant. As a rule, when there is copious effusion, but not enough to completely fill the chest, the upper line of dulness is a curved one and its shape approximates to that of the letter S, the highest part of the curve being over the scapula and in the axilla, the lowest part near the spine. Behind, then, there is commonly a band of resonance, more or less impaired, in the interscapular region, and over the adjacent portion of the scapula, including the suprascapular fossa.

In front, whenever effusion is considerable in amount, the upper line of dulness crosses the middle line, descending from the neighbourhood of the apex of the lung to join the dulness yielded by the more or less displaced heart (see Fig. 84).

After removal of the fluid, either by paracentesis or by absorption, the infra-axillary region is usually the last place where dulness may be obtained.

It is important to remember that the extent and degree of dulness is not a measure of the quantity of fluid present; a small empyema, for example, may coexist with extensive and absolute dulness; or, on the other hand, resonance may be only slightly or moderately impaired when there is a considerable amount of effusion; also, after removal of serum or pus, the dulness sometimes remains as great as ever.

(*c.*) In pneumothorax when the pleural cavity is greatly distended.

(*d.*) Over tumours or thick false membranes.

4. Rarely in extreme emphysema.

5. When the lung adjacent to the chest-wall is partially or completely impermeable to air, as when collapsed from any cause, or when it is the seat of inflammatory or tubercular infiltrations, of hæmorrhagic infarcts, of abscesses or new growths.

The most common causes of impaired resonance are,—solidification of lung tissue and liquid in the pleural cavity.

Metallic Ring or Amphoric Resonance.—A high-pitched hollow metallic sound, whose fundamental tone is accompanied by overtones which give it a prolonged metallic echo, is met with over large superficial air spaces with smooth walls, either completely closed or communicating with a bronchial tube by means of a small opening. Examples: pneumothorax; large pulmonary cavities.

Cracked-pot Sound (*Bruit de pot fêlé*). — This may be roughly imitated by clasping the hands loosely together and striking the back of one of them upon the knee, the enclosed air being suddenly expelled through a small opening. It is produced in health sometimes over the yielding chest of a healthy screaming infant; in disease (1.) over a superficial cavity connected with a bronchus by a narrow opening, as at a phthisical apex or in pneumothorax. (2.) Sometimes over relaxed lung-tissue, as at the upper limit of a pleuritic effusion or over a partially consolidated lung. In children especially it is by no means uncommon to obtain a cracked-pot sound below the clavicle on the side of a pleural effusion.

As a rule, a heavy percussion stroke is necessary to bring out the sound; it should be delivered during expiration, after the patient has taken a full inspiration, and while his mouth is open.

Resistance.—The feeling of resistance varies inversely with the

compressibility of the part percussed; for example, it is much less over the yielding thorax of the child than over the rigid chest of advanced life. It is greater over solids and liquids than over air-containing tissues; thus resistance is increased in solidification of the lung, still more so over a liquid pleural effusion, and most of all over an intra-thoracic tumour.

It is diminished in moderate distension of the lung or pleura, as in pneumothorax and emphysema, but if the distension is extreme, there is an increase of resistance to percussion.

The value of this sign is very great; it enters largely, though often unconsciously, into our conception of the differences observed between the results of percussion over a healthy and a diseased part of the chest.

AUSCULTATION.

Auscultation is the act of listening to the sounds produced in the body by means of the application of the ear directly or indirectly to the body surface. It implies the voluntary effort of bringing the ear into some sort of contact with the surface, and includes everything heard—whether the sounds be produced in the respiratory organs, in the heart and large vessels, in the alimentary canal, or in connection with any other organ.

In **auscultating the respiratory organs**, we listen for the normal and abnormal sounds produced by breathing, speaking and coughing. In immediate auscultation the ear is applied directly to the chest, in mediate auscultation through the medium of the stethoscope. Both methods have their advantages. By listening directly to the chest, a rapid estimation can be made of the condition of a large portion of lung, and for the dorsal region this is the only method permissible when the patient is too feeble to be held up for more than a few seconds; the method is also especially suitable for the examination of the back of a young child. The breath sounds are heard louder than with a stethoscope, but many delicate sounds may be lost. The accurate localisation of sounds, too, is difficult, and examination of the apices is obviously unsatisfactory.

Rules to be Observed in Stethoscopic Examination.—1. In all cases, when practicable, the stethoscope should be applied directly to the skin.

2. Great care should be taken that every part of the circumference of the conical end is in accurate contact with the skin; this is best ensured by first grasping the lower end of the stethoscope with the finger and thumb (usually of the left hand), and holding it evenly, gently, but with sufficient firmness against the chest-wall, until the ear has been carefully adjusted to the other end. When this is accom-

plished, and the observer feels that the stethoscope is well balanced, and that both ends are accurately adjusted, the finger and thumb should be removed from the stethoscope, and used with the other fingers to separate the clothes (if the chest is not stripped), and so prevent rustling noises through contact with the stethoscope.

3. Each part of the chest—front, axillæ, back—is to be carefully examined and compared. In comparing the two sides of the chest, the stethoscope should be placed on symmetrical spots.

Auscultation of the Healthy Chest.—It is most important that the student, before listening to diseased chests, should become perfectly familiar with the normal sounds heard on listening over the respiratory organs. They are those of respiration, of phonation (produced at the rima glottidis by the vocal cords), and of articulation (produced only in the cavity of the mouth). The student should therefore listen while the person breathes, while he speaks in his natural voice, and while he whispers (which gives the simple articulatory sound). And the following exercise may be usefully undertaken at home with the help of a fellow-student:—

Exercise 1.—Apply the stethoscope to one side of the larynx, and observe the inspiratory and expiratory sound during quiet and during forced breathing, noting the length of each, and comparing it with that of each respiratory *movement;* the interval between the sounds; their pitch and peculiar hollow quality.

2. Apply the stethoscope or the ear directly to the left infra-scapular region, and carefully observe the marked difference between the breath sounds here and over the larynx.

It is very desirable that the student should auscultate the places mentioned daily during the first week or two of his beginning the study of auscultation, and it is well to write out, and more than once, his own description of what he hears, and compare it with that of a fellow-student, and afterwards with the details given below. Too much stress cannot be laid on the importance of this exercise, for the breath sounds as heard over the windpipe and over the spongy lung-tissue form **two great types** under which all morbid breath sounds may be classed. Once mastered and retained as standards in the mind, there are but few future auscultatory troubles for the student.

3. Having studied these types, he should listen to other parts of the chest, and observe any minor modification of the breath sounds, especially comparing the right with the left apex, and the sounds heard over the manubrium and in the inter-scapular region with those heard in other parts of the chest.

4. The normal sounds of vocalisation and articulation should now be studied by listening over the larynx and over the infra-scapular

region on the left side—(1) when the person speaks in his natural voice, and (2) when he whispers.

Breath Sounds in Health.—On auscultating the respiratory organs, two sounds or murmurs are heard, one accompanying the act of inspiration, the other that of expiration. A marked contrast exists between the breath sounds heard over the larynx and trachea, and those heard over the chest-wall covering the vesicular tissue of the lung.

The Laryngeal or Tracheal sounds are blowing and harsh, begin and end abruptly, are of about the same duration, and are separated by a very short but distinct pause. The expiratory is softer than the inspiratory murmur, and during exaggerated breathing exceeds it in length. Each lasts as long as the inspiratory and expiratory movement that produces it. They may be imitated by breathing in and out through the lips, pushed forwards as in pronouncing "*chur*." On listening over the manubrium, and in the inter-scapular region at the level of the bifurcation of the trachea, weaker sounds, but sounds identical in quality, are heard in many persons; hence this type of breathing is called "**bronchial.**"

The sounds heard over the healthy lung, called "**vesicular,**" are softer and lower in pitch than the laryngeal, and lack its hollow reverberating character. There is no interval between them; they form a continuous breezy murmur, like the sighing of wind through the leaves of a tree. The expiratory part of the murmur is feebler, lower in pitch, and one-fourth to one-fifth shorter than the inspiratory, and is often quite inaudible. The inspiratory begins and ends with the inspiratory movement of the chest; the expiratory is only heard at the very commencement of the expiratory act. A careful distinction should be drawn between the sound of ordinary tranquil respiration and that of forced or deep breathing. The latter tends to lose the vesicular character, and often approximates to harsh breathing.

Healthy Varieties.—The qualities of the vesicular murmur vary slightly in different parts of the chest. Generally speaking, it is louder over the more resonant parts—thus in front than behind, above than below. At the apices the vesicular quality is less marked, the pitch is higher, and the expiratory sound is longer than over the infra-scapular region; and these peculiarities are more noticeable at the right than at the left apex, the breath sound in the right infra-clavicular region being somewhat higher in pitch, and expiration longer than in the left infra-clavicular region, whereas the inspiratory murmur is nearly always louder at the left apex.

In infancy the respiratory murmur is louder and less breezy, and is called "*puerile;*" in old age it is feebler than in the adult. The expiratory sound is also longer at the two extremes of life.

Mechanism.—The laryngeal breath sounds are caused by fluid veins produced at the glottis by the passage of the air through the narrow space between the vocal cords into the wider spaces above and below. The vesicular murmur has a double mechanism; it is mainly produced (and its expiratory portion probably entirely) at the rima glottidis, the laryngeal character being lost by conduction through the spongy texture of the lung. Its inspiratory portion is also made up of the innumerable minute murmurs evoked by the passage of air from the end of each bronchiole into its infundibulum.

Auscultation of the Voice in Health.—The voice in health is made up of two elements, namely, phonation and articulation; the former is produced by the vibrations of the vocal cords, the latter in the mouth by the movements of the tongue, lips, and palate.

Phonation.—On listening with a stethoscope to the larynx of a healthy person during the act of singing or speaking aloud, the musical part of the voice is heard with almost painful intensity; a similar but less distinct voice sound is heard over the first piece of the sternum or behind between the scapular spines; but over the rest of the chest-wall which covers healthy lung-tissue a mere humming or buzzing is audible.

Articulation.—The articulate voice, best heard when the patient whispers, is distinct on listening over the larynx; also often over the situation of the chief bronchi, although there it is usually difficult to identify the words whispered. But over the chief part of the pulmonary surface articulation is completely lost.

Auscultation in Disease.—We meet with (1) modifications of the normal breath sounds; (2) modifications of the normal sounds of phonation and of articulation; (3) adventitious sounds. Most of the phenomena enumerated below may be studied by the examination of a case of **pleuritic effusion** and a case of **chronic phthisis.** In the former, let the student note the feebleness of the breath sounds over the effusion, with their loudness over the opposite lung. In the latter, let him compare the hollow bronchial breath sound with the normal laryngeal breathing. In both let him examine carefully for bronchophony and pectoriloquy, and for adventitious sounds.

Modifications of the Vesicular Type.—**1. Weakening or Suppression of the Vesicular Murmur.**—This may occur: (1.) From feebleness of the movements of the thorax, as on the side of a pleuritic stitch. (2.) From obstruction of the air passages, either (*a*) from within, as in stricture of the trachea, in bronchitis, and sometimes in cases of pulmonary consolidation, owing to blocking of the bronchi with secretion; or (*b*) from without, as when a bronchus is compressed by an aneurysm or a new growth. (3.) When there are fluids or solids between the lung and chest-wall; thus in pleuritic effusion, or when

there are massive adhesions, also sometimes in pneumo-thorax, when no open communication exists between the lung and pleural cavity.

2. **Harsh, Exaggerated, or Puerile Breathing.**—This type of breathing is heard over the chest of a healthy child. Both inspiration and expiration are louder and of harsher quality than in the adult. When the result of thoracic disease, sometimes inspiration, sometimes expiration, is the harsher of the two, and expiration may be prolonged slightly, but there is no lengthening of the pause. The lengthening of expiration in harsh breathing is usually more apparent than real—that is, the ordinary expiratory sound of normal respiration becomes louder, and is therefore more manifest.

Harsh breathing may be heard over healthy lung when another portion of the lung or when its fellow is compressed, solidified, or otherwise disabled; thus, in a case of pleuritic effusion, harsh breath sounds are usually audible over the unaffected side. It is also very common over minor or moderate degrees of consolidation, as at the apex in phthisis.

3. **Prolongation of Expiration.**—This is a feature of bronchial breathing, but sometimes it is the chief or the only modification, as when the elasticity of the lung is diminished in emphysema, or when there is an obstruction to the exit of air, as occurs in bronchitis. It is often one of the first indications of a commencing consolidation at the apex.

4. **Jerky or Wavy Breathing,** in which inspiration is interrupted two or three times, is heard over the whole lung: (1) sometimes in hysteria; (2) in painful affections of the respiratory muscles, causing them to act irregularly.

Locally it is of more importance, as when met with at the apex, when inspiration has sometimes a "cogged-wheel rhythm," a sign often of incipient phthisis. It is thought to be produced by obstructions in the finer bronchioles, and is probably of the nature of a rhonchus.

5. **Deferred Inspiration.**—Inspiration is said to be deferred when the inspiratory movement of the chest is felt before any sound is heard. This occurs in emphysema and in laryngeal obstruction.

6. **Broncho-Vesicular or Transitional Breathing.**—Sometimes it is very difficult to say whether the breath sounds belong to the vesicular or to the bronchial type; for example, at a phthisical apex, and in some cases of emphysema, the breath sounds are harsh, divided, and expiration is as long as inspiration; they thus approach the bronchial type, but the peculiar "*ch*" quality of true glottic breathing is either absent or doubtful. Skoda classed such sounds as "indeterminate," but it is better for the student, when in doubt as to their nature, to describe them as accurately as he can—the quality, the pause, and the relative

length of the inspiratory and expiratory portions, rather than to merely give them a definite name.

Modifications of the Normal Laryngeal or "Bronchial" Type.—Bronchial breathing, similar to healthy laryngeal or tracheal breathing, is heard over the chest, where normally the vesicular is audible, when the lung-tissue has ceased to contain air, being consolidated as in pneumonia, collapsed or compressed as in cases of pleuritic effusion. In these cases the bronchial tubes are surrounded by solid tissue, and hence the glottic sounds are well conducted, while any sounds which originate in the parenchyma are suppressed.

According to the character of the sounds heard, bronchial breathing is described as **tubular**, **blowing**, or **cavernous**.

Tubular most nearly approaches to the normal breathing heard over the larynx. It is, however, higher in pitch, it conveys the sensation of air being drawn into and puffed out of a narrow metallic tubular space, which appears to be immediately beneath the part examined. It is heard to perfection over a hepatised lobe in pneumonia, and its metallic quality is highly developed. In **blowing** breathing there is less concentration, the sounds appear to be produced at a distance and in a wider tube. In **cavernous** breathing the ear gets the impression of a still larger space, the inspiratory and expiratory sounds are both of a hollow whiffing character, and are lower pitched, especially the expiratory, than in tubular breathing. Mostly heard over cavities in the lung, it may also be produced when there is solid tissue between the root of the lung and the surface.

These modifications of the normal glottic breath sounds are produced by varying degrees of solidification or excavation of lung-tissue. The precise amount of change in the lung structure cannot be determined by these variations. The important information derived from the presence of the bronchial type, whether of tubular, blowing, or cavernous quality, is that the lung parenchyma no longer admits air. The bronchial breath sound is recognised, it cannot be too often repeated, by its **peculiar quality**, and not because it is louder than the vesicular murmur; indeed, perfect tubular breathing of extreme weakness may often be distinguished over an effusion into the pleural cavity.

Auscultation of the Voice in Disease.—Vocal Resonance may be defined to be the voice as it is heard on applying the ear, directly or indirectly, by means of a stethoscope, to the surface of the chest.

Weakness or Absence of Vocal Resonance is met with in cases of obstruction or compression of the bronchial tubes, or when fluid or morbid tissue intervenes between the lung and the thoracic wall; thus it is found in pleuritic effusion, in pneumothorax, and in cases where the pleural cavity is obliterated by dense false membranes.

Intensification of the Vocal Resonance or Bronchophony.—It is impossible to distinguish between a simple increase of the vocal resonance and bronchophony; hence they are here classed together, and bronchophony is defined as an increased distinctness or clearness of vocal resonance at the surface of the chest. The musical tones formed at the glottis reach the ear not as a vague humming, but with distinctness and clearness, and in these respects are similar to the sounds heard over the windpipe in the neck.

Bronchophony, as already mentioned, may often be heard over the bifurcation of the bronchi, and sometimes beneath the right clavicle, especially in women and children. When present at other parts of the chest, it is pathological and has a similar significance, speaking broadly, to bronchial breathing. Thus it occurs (1) in consolidation of the lung, as from collapse, pneumonia, phthisis, hæmorrhagic infarct and tumours; (2) over excavations in the lung or dilatations of the bronchi, provided that the tissue around such cavities is solid; (3) sometimes in cases of extreme emphysema. But it is to be noted that if in cases of consolidation or of excavation of the lung, the bronchial channel be obstructed between the larynx and the conducting part of the lung, as from a plug of secretion, the production of bronchophony is interfered with, and the voice sound is enfeebled or annulled.

Pectoriloquy is a term best restricted to the transmission of the articulate utterance of a patient directly to the ear or along the stethoscope of the auscultator. It refers to articulation, bronchophony to phonation. A pure articulatory sound is best obtained by getting the patient to whisper. Then if each syllable sounds distinctly, and as if produced in the tube of the stethoscope, pectoriloquy is said to be present. Pectoriloquy is most frequently met with over superficial cavities, which freely communicate with bronchial tubes. Like bronchophony, it is sometimes present over solid portions of lung, and is therefore not a certain sign of a cavity. This physical sign is also often to be recognised over a pleuritic effusion, and perhaps more frequently when the effusion is serous than when it is purulent.

Ægophony is a peculiar modification of bronchophony, in which the voice resembles the squeaking of a Punch and Judy exhibitor or the bleating of a goat. The voice is higher pitched and shriller than that coming from the patient's mouth, and has often a tremulous or jerky character. It is best heard in cases of moderate pleural effusion on applying the ear near the angle of the scapula; but true ægophony is not a common phenomenon. The name, however, is often given to ordinary bronchophony possessing a slightly nasal twang; this might be termed **nasal bronchophony.**

Resonance of the cough or cry may be of value in the absence of

other signs. Thus in auscultating the chest of a restless screaming infant, when the breath sounds, for obvious reasons, are difficult to appreciate, a ringing bronchophonic cry is suggestive of hepatisation of the underlying lobe.

Adventitious Sounds.—In most diseases of the lungs and pleuræ, at some part of their course, the respiratory murmur is accompanied by certain adventitious sounds, which are known as rhonchi, râles and friction sounds. These may be grouped as follows :—

Rhonchi or dry sounds.

1. Sonorous or low-pitched rhonchi.
2. Sibilant or high-pitched rhonchi.

Râles or moist sounds.

1. Crepitation.
2. Bubbling râles.
   - (1.) Simple. { Small bubbling or subcrepitant râles. / Medium bubbling or submucous râles. / Large bubbling or mucous râles.
   - (2.) Metallic or consonating. { Small metallic or crackling râles. / Medium „ „ / Large „ „ } Called also "dry and moist crackling."

"Laennec's dry crepitant râles with large bubbles."

Pleuritic friction.

Rhonchi, sometimes called dry râles, are musical continuous sounds, which accompany inspiration and expiration. They may completely obscure both breath sounds, and may be audible away from the patient. They are classed as sonorous and sibilant. The former are low-pitched, cooing or snoring sounds; the latter are high-pitched, and of whistling or hissing character. They are produced by the passage of air through a bronchial tube which is narrowed at some point, the constriction being due (1) usually to the presence of viscid mucus; (2) to swelling of the bronchial mucous membrane; or (3) to contraction or compression of a bronchial tube, the first occurring in asthma, the last sometimes in aortic aneurysm.

Crepitation.—This term is applied to very fine sharp moist sounds heard during inspiration, usually towards its termination, but not during expiration. They may be imitated by rubbing a lock of hair between the fingers close to the ear, or by rubbing the moistened thumb against the forefinger. Crepitations are uniform in size, and are unaffected by coughing. They are typically met with in the first stage of pneumonia; also over œdematous or collapsed lung (see "Collapse râle," p. 164). The last condition indicates their nature, namely, that they are due to the sudden expansion of alveolar walls glued together by viscid secretion.

This râle has been called "vesicular," and it gives us the important information that air can still enter the air cells. Sometimes the bursting of innumerable small bubbles produces sounds identical with those of true crepitation.

Simple Bubbling, or mucous râles, of various size, are produced in bronchial tubes or in cavities in the lung, by the passage of air through mucus, serum, blood, or other liquid. They are irregular in size, and are modified by coughing and by expectoration, differing in these respects from the crepitant râles as just described. Their size depends on the quantity and quality of the fluid, and to some extent on the capacity of the space in which they originate, although it must be borne in mind that small bubbles may be formed in large tubes. The smallest sized bubbling, sometimes called the subcrepitant râle, occurs in capillary bronchitis; the largest is produced in the trachea and bronchi, and indicates an accumulation of fluid owing to failure of vital powers; hence it is popularly known as the "death rattle." It is especially noticeable when death takes place during coma, as in apoplexy, and the last stage of many pulmonary affections.

Metallic Râles.—These are bubbling râles of various sizes, which have a peculiar metallic character. They are sometimes called "consonating," owing to their clearness and resonance, and the term may be taken to include the "dry and moist crackling" spoken of by some writers, and which are so frequently met with at a phthisical apex. Besides indicating the presence of liquid in bronchial tubes or in pulmonary cavities, they also suggest that the latter are surrounded by solid tissue. A variety called "clicking," from the character of the sound, is produced during inspiration only, and is common at the apex in phthisis.

Dry Crepitant Râle with Large Bubbles.—This term was given by Laennec to a sound which conveys "the sensation of air distending dry and very unequally dilated pulmonary vesicles." It is a rare phenomenon, and is practically limited to extreme cases of emphysema. It resembles a high-pitched bubbling sound, is persistent, accompanies inspiration, and is best heard in the infra-axillary regions.

Dr. Steell has drawn attention to a crackling sound closely related to the above râle, and "audible in the neighbourhood of the pulmonary artery over the border of the left lung. Usually it is heard only during inspiration, but it may be present to some degree during expiration, and may temporarily assume cardiac rhythm." He thinks that the sound is produced by an emphysematous state of the edge of the left lung, and that its "intermittence and variableness may be explained by varying conditions of lung compression by the heart," the heart in these cases being usually enlarged.

Friction Sounds.—In health the gliding of one pleural surface over the other is silently performed, but any unevenness of the opposed surfaces, or the deposit of morbid material between them, is liable to produce friction sounds. These vary from the slightest grazing sound to loud creaking or crackling. They are commonly restricted to a small area of the lower half of the chest in the lateral region, or near the lower angle of the scapula. They may accompany both inspiration and expiration, but as a rule they are present only towards the end of inspiration. They are less continuous than râles, often occur in a series of jerks, are uninfluenced by coughing, and are strengthened by a deep inspiration, and sometimes by pressure of the stethoscope on the intercostal space. They are usually associated with a stitch-like pain, with enfeebled breath sounds, and with diminished movement of the affected side. The friction sound disappears before the advancing fluid, but is again audible, and often with greater intensity, during its subsidence. The loudness of the sound bears no relation to the amount or condition of the exuded lymph. Besides lymph, the sound may be caused by the presence of miliary tubercles, of false membranes, and even by increased vascularity and dryness of the pleural surfaces. Friction sounds are occasionally produced by the movements of the heart, and are then audible when the patient holds his breath. Extensive friction sound over the right side may occasionally be produced by inflammatory products between the liver and diaphragm (perihepatitis), and more rarely also on the left side from perisplenitis.

Amphoric Echo.—This is a peculiar metallic sound, similar to that produced by blowing into an empty bottle or jug. For its production a large air-containing cavity is necessary, but the presence of liquid is not essential. It may accompany the breath sounds, the voice, cough and râles, or even the cardiac sounds. It occurs in cases of large cavities in the lungs, and in pneumothorax; in the latter case the amphoric quality is given to the bronchial breath sounds by their passage from the collapsed lung through the pleural air cavity, which may be quite closed. This echo too sometimes accompanies the cardiac sounds, and in such a case may be generated by a gas-distended stomach.

Metallic Tinkling is a clear, ringing, highly metallic single sound, which is best imitated by letting drops of water fall on the surface of a little water in a decanter. It occurs along with or alternates with amphoric echo, and its explanation and associations are the same.

The Bell Sound.—If the stethoscope be applied over a pneumothorax or other large air-containing cavity while the side is percussed by two coins, a sound is produced which closely resembles that heard at a distance from an anvil struck with a hammer. When this bell or

anvil-like sound is present, the limits of the air-containing cavity may sometimes be accurately mapped out by percussion of the coins in different places while the stethoscope is stationary; for the bell sound is lost when the coins are percussed outside the boundaries of the air space.

Fallacious Auscultatory Sounds.—1. A dry rubbing sound, confined usually to inspiration, is occasionally heard in the supraspinous fossa near the shoulder-joint, where it doubtless originates.

2. A rumbling sound is not uncommon. It is produced by muscular contraction, and is usually continuous.

3. Conduction of râles across the spine. This may be observed in pneumonia of the lower lobe, the crepitations being heard on the posterior aspect of the unaffected side.

4. *Collapse Râle.*—Fine crepitant sounds are sometimes heard at the base of the lung posteriorly, when a person with healthy lungs sits up in bed and takes a deep inspiration, but they readily disappear after a few inspirations. It is necessary for the young student to be on his guard against confusing this collapse râle at the bases of a bed-ridden patient with crepitation on the one hand and friction on the other. Occasionally, too, this râle occurs at the apex in cases of typhoid fever, and, if not recognised as a "collapse râle," might lead the observer to regard the case as one of tuberculosis.

Succussion—The Hippocratic Succussion Sound.—A distinct splashing sound may be produced in a large cavity containing air and liquid by giving the patient a smart shake, while the ear is applied to the surface of the trunk; sometimes the succussion sound is loud enough to be audible to attentive bystanders, or even to the patient himself. As a rule, the sign indicates the existence of a hydro-pneumothorax. If heard on the left side of the chest, the possibility of the sound being generated by a dilated stomach must be considered.

A feeble succussion sound is sometimes audible over a cavity in the lung during the act of coughing, the cough propelling air into the cavity and thus setting up gurgling or splashing râles, and in cases where a cavity furnishes no characteristic bronchial breath sound, a succussion sound or succussion râles may be of value in diagnosis.

THE SPUTUM.

In health the expectoration is very small in amount; it consists of a colourless transparent sticky fluid. In various diseased conditions the amount is greatly increased and the character altered.

Sputum may be divided into the following varieties:—

1. **Mucous.**—The expectoration is clear, viscid, tenacious and trans-

parent. This is the variety met with in the early stage of bronchitis. It consists chiefly of mucus and a few cells. Mucous expectoration is generally followed by a more opaque variety at a later stage of the illness.

2. **Purulent.**—The expectoration consists chiefly of pus, and is greenish-yellow in colour. This variety is met with in cases of empyema bursting into the lung, in pulmonary abscess and in tubercular cavities.

3. **Muco-purulent.**—This is the most common variety. Its characters are intermediate between mucous and purulent expectoration; it is really a mixture of transparent mucous and greenish-opaque purulent sputum.

Mucous, muco-purulent and purulent sputa are met with in the successive stages of bronchitis; mucous during the acute stage, muco-purulent and purulent in the advanced stages, or at the commencement of convalescence.

4. **Serous.**—The expectoration is a thin frothy-looking fluid, which consists principally of serum. This variety occurs in œdema of the lungs, and sometimes during or after the operation of paracentesis of the chest. In the latter case the sero-albuminous expectoration may be very copious, and the associated cough very troublesome.

5. **Sanguineous.**—Blood may be expectorated in the form of streaks or clots, or it may be intimately mixed with the sputum. When sudden and profuse hæmorrhage into the air passages takes place, large quantities of pure blood may be coughed up.

The **Quantity** of sputum is sometimes very great. Thus in bronchiectasis large quantities may be brought up at one time. Pus passing suddenly into the bronchial tubes gives rise to profuse purulent expectoration, as in cases of empyema bursting into the lung, or in cases of pulmonary abscess, tubercular cavity, abscess of the liver or mediastinum discharging their contents into the bronchial tubes.

The Odour.—In gangrene of the lung the expectoration and breath have a peculiar, intensely pungent and fœtid smell. The sputum is usually very fœtid in bronchiectasis, bronchorrhœa, and often when derived from pulmonary cavities or abscesses.

Casts.—In plastic bronchitis, and occasionally in acute croupous pneumonia, fibrinous casts of the small bronchial tubes are found in the sputum.

In diphtheria fibrinous shreds or pieces of **membrane** are sometimes expectorated.

In hydatid of the lung and in hydatid of the liver, which has burst into the bronchial tubes, **daughter cysts**, not unlike empty gooseberry skins, are sometimes coughed up, while scolices and hydatid **hooklets** may be found in the sputum on microscopical examination.

4. Ulcerative affections of the larynx, trachea, or bronchi.

5. In pneumonia the expectoration at first may be scanty and colourless, but it soon assumes a rusty colour, which is characteristic of the disease. At this stage the sputum is almost airless and very tenacious, and adheres firmly to the vessel. The colour is due to dissolved hæmoglobin, and to the presence of a small number of red corpuscles. Sometimes the sputum is grass green, probably owing to the conversion of hæmoglobin into bilirubin, and afterwards into biliverdin. At a later stage the expectoration increases in amount; it becomes more watery and yellowish or greenish in colour.

6. Hæmoptysis sometimes occurs in blood diseases, as purpura, or in diseases in which there is a marked hæmorrhagic tendency, such as hæmorrhagic small-pox.

7. In young healthy persons, hæmoptysis occasionally occurs, and the patient may recover completely without the appearance of any subsequent symptoms of lung disease.

8. Since the days of Hippocrates, a relation has been supposed to exist between hæmoptysis and menstruation, and a few rare cases have been recorded on good authority of vicarious menstruation in this form.

9. Sir Andrew Clark has called attention to a form of recurring hæmoptysis in arthritic subjects. The patients are over fifty years of age; the disease is not followed by pulmonary changes, and rarely ends fatally.

10. Blood from the nose may trickle down the throat and be expectorated, and so simulate hæmoptysis. Also blood in the expectoration is sometimes due to hæmorrhage from the gums.

In chronic pneumonia with destruction of lung-tissue, and in certain cases of aneurysm, the intimate admixture of blood and sputum gives rise to a dark expectoration resembling prune juice.

Sometimes there is considerable difficulty in deciding whether blood, especially if large in amount, is coughed up or vomited, *i.e.*, whether the case is one of hæmoptysis or hæmatemesis. Blood from the lungs is usually brighter red and more frothy than that from the stomach. The latter is often mixed up with partially digested food, and is usually acid in reaction, whereas expectorated blood is alkaline. But an examination of the chest and abdomen, as well as a careful consideration of the history and other aspects of the case, are often necessary before a diagnosis can be established.

Tubercle Bacilli.—The examination of the sputum for the tubercle bacillus in cases of suspected tubercular disease is of the greatest importance in practical medicine. By the presence of these bacilli in the sputum, the diagnosis of tubercular disease can be definitely settled in an early stage of the disease, when the physical signs and symptoms

would not justify a positive diagnosis—*i.e.*, at a time when the prospects of a cure by treatment are the greatest. Hence the student should become thoroughly well acquainted with the method of examination for tubercle bacilli.

Fränkel-Gabbett's is one of the most convenient and reliable methods. By means of a pair of forceps, a small piece of sputum is taken from a vessel containing the expectoration and placed on a cover-glass, which has previously been well cleaned. It is best to take a bit of the thickest and most purulent portion of the sputum. The sputum is spread over the cover-glass, and then a second well-cleaned cover-glass is placed on the first. The two are rubbed together so as to obtain a thin uniform layer of sputum, spread out between the cover-glasses. They are then separated, and a thin layer of sputum is thus obtained on each cover-glass. The cover-glasses are then dried over the flame of a spirit-lamp or Bunsen's burner, the surface on which the sputum is spread being kept upwards. When quite dry they are passed rapidly three times through the flame of a Bunsen's burner or spirit-lamp. They are then floated on a magenta solution in a watch-glass or capsule, the side on which the sputum is fixed being in contact with the fluid. The strength of the solution is as follows :—

Magenta	1 gramme.
Absolute alcohol	10 cc.
5 per cent. solution of carbolic acid . .	90 cc.

The solution is warmed until vapour is given off freely, but it must not be heated to the boiling-point.

The cover-glasses should remain in this solution for about four minutes, and should then be washed in water for a second or two. They are then floated (the surface on which the sputum is spread being downwards) in a solution of methyl-blue and sulphuric acid, or a little of this solution may be dropped on to the sputum-covered surface of the glass slip.

The methyl-blue and acid solution has the following composition :—

Methyl-blue	2 grammes.
25 per cent. solution of sulphuric acid .	100 cc.

After staining for one minute, the blue solution is washed away thoroughly with water ; the cover-glass is dried and mounted in Canada balsam.

Precautions.—1. It is desirable not to place too large a piece of sputum between the cover-glasses ; otherwise almost the whole of the sputum is squeezed out when the cover-glasses are rubbed together.

2. In drying the cover-glasses, it is well to hold them in the fingers, for if held by forceps there is a danger of the specimens getting too hot and becoming charred.

3. When the cover-glasses are placed on the carbol-magenta solution, it is well to see that they either float perfectly or sink to the bottom. If floating with half the cover-glass above the surface and half below, a line of deep red stain is formed on the cover-glass, which is difficult to remove.

4. After staining in the methyl-blue solution, the cover-glasses should be thoroughly well washed in water; but before mounting in Canada balsam they must be perfectly dry.

5. In mounting the specimen, it is well to avoid using too much Canada balsam, for an excess of balsam surrounds the cover-glass as a raised rim, and is liable to be smeared on the objective of the microscope. Xylol balsam is very useful for mounting the specimen.

This method of staining is exceedingly convenient, and can be carried out in a few minutes.

In order to avoid any risk of self-infection from the tubercular sputum, the fingers should be thoroughly cleansed as soon as the specimens are prepared; also it is advisable to burn the sputum when it is no longer required for examination.

By this method of staining, at first all parts of the cover-glass preparation are stained red with the carbol-magenta. The acid in the second solution removes the red stain from everything except the tubercle bacilli, other structures being blue from the action of the methylene blue. No other bacilli stain red in this way except those of leprosy. Under the microscope, tubercle bacilli appear as small slender rods 1.5 μ–3.5 μ in length, they are slightly curved, and sometimes contain clear spaces which do not stain in the same manner as the rest of the bacillus; hence tubercle bacilli often have a beaded appearance. Their presence is a certain indication of tubercular disease. Their absence, however, does not prove the absence of tubercular disease. It sometimes happens that tubercle bacilli are only discovered in the sputum after repeated, prolonged and careful examinations.

It is well to remember that the presence of tubercle bacilli does not necessarily indicate a fatal termination of the disease. Cases of complete recovery sometimes occur, even after tubercle bacilli have been found in the sputum in great numbers. Moreover, the number of bacilli present in any specimen cannot be regarded as an indication of the degree of severity of the disease.

Tubercle bacilli are present in all forms of acute and chronic pulmonary tuberculosis, but in miliary tuberculosis of the lungs, according to von Jaksch, they are always absent from the sputum, which resembles that of acute catarrh.

Another method much in use is that of **Ziehl-Neelsen.** In this method the specimens are prepared and stained in the carbol-magenta

solution, as described above, but the cover-glasses are then placed in a 25 per cent. solution of sulphuric acid for a few seconds, until the red colour is changed to a brownish-yellow, which occurs very rapidly. They are then washed well in water, and stained with a watery solution of methyl-blue.

Nitric acid, 1 of strong acid to 3 parts of water, or hydrochloric acid 33 per cent. solution, is used, in place of sulphuric acid in other methods.

Biedert's Method.—A useful method is described by Biedert for detecting tubercle bacilli when these organisms are present only in small numbers in the sputum. [The method is of great service in examining urine for tubercle bacilli.] One tablespoonful of the sputum is mixed thoroughly with two tablespoonfuls of water and four drops of caustic soda solution added. If the mixture is very thick, eight drops may be added, but too much caustic soda must be avoided, otherwise

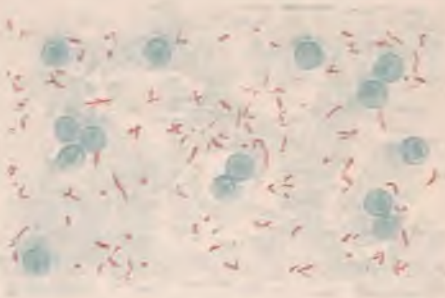


FIG. 55.—Tubercle Bacilli stained by the Ziehl-Neelsen Method. (*v. Jaksch.*)

the staining of the bacilli is impaired. The mixture is then boiled and stirred with a glass rod, until a moderately thin limpid fluid is obtained. Four to six tablespoonfuls of water are then gradually added. The fluid is placed in a urine glass having a conical bottom, and allowed to stand two days—not longer. The fluid is then decanted until a sediment of about $\frac{1}{2}$–$\frac{3}{4}$ cm. only remains, and this is examined for tubercle bacilli in the usual way. If there is any difficulty in causing the sediment to remain adherent to the cover-glass, a small portion of the original sputum or a little egg albumen may be added. It is well to allow the cover-glass to remain in the magenta solution (Gabbett's method) a little longer than usual when staining the sediment.

Elastic Tissue.—Osler gives the following method, which was demonstrated to him by Sir Andrew Clark. It depends upon the fact "that in almost all instances, if the sputum is spread in a sufficiently thin layer, the fragments of elastic tissue can be seen with the naked eye.

The thick purulent portions are placed upon a glass plate 15 by 15 centimetres, and flattened into a thin layer by a second plate 10 by 10 centimetres. In this compressed greyish layer between the glass slips any fragments of elastic tissue show on a black background as greyish-yellow spots, and can either be examined at once under a low power, or the uppermost piece of glass is slid along until the fragment is exposed, when it is picked out and placed upon an ordinary microscopic slide. Fragments of bread and collections of small globules may also present an opaque white appearance, but, with a little practice, they can readily be recognised. Fragments of epithelium from the tongue infiltrated with micrococci are still more deceptive, but the microscope at once shows the difference." Instead of the glass plates, two large microscopical slides may be used.

In order to detect elastic fibres, Fenwick boils the sputum with caustic soda. The sputum is mixed with an equal quantity of caustic soda solution (20 grains to the ounce of water). The mixture is boiled in a glass beaker, being well stirred with a glass rod. As soon as it boils, it is poured into a conical glass, and four or five times the amount of cold distilled water is added. Any lung-tissue sinks to the bottom, and may then be removed by a pipette and examined microscopically.

Elastic fibres occur in tubercular lung disease, pulmonary abscess, and rarely in gangrene. Their presence indicates destruction of lung-tissue. It is said that in gangrene the elastic fibres are often destroyed by a ferment formed in the gangrenous parts.

Elastic fibres are slightly curved, generally exhibit a double contour, and, when coming from the lung alveoli, often show a branched alveolar arrangement.

V. Jaksch points out that elastic tissue may be introduced with food, and so find its way into sputum. Hence the mouth ought to be washed out carefully after food, and the sputa discharged at meal-times ought to be separated from those to be examined for lung-tissue. "It is only when the bundles of elastic fibres display the alveolar arrangement that we can be certain of their origin in the pulmonary alveoli, and it is only then that they possess any sure diagnostic significance" (v. Jaksch).

Curschmann's Spirals and Charcot-Leyden Crystals.—In cases of asthma the sputum often contains yellowish and greyish masses. The former consist of degenerated pus corpuscles with octahedral crystals. These crystals are identical chemically with Charcot's crystals, which are found in semen, in bone-marrow, and in the spleen in leucocythæmia. The grey plugs consist of spiral threads, some of which can be recognised by the naked eye, others only by the microscope. These spirals are casts of the smallest bronchi; they often present a brilliant

band in the middle of the spiral. The spirals and crystals are expectorated during the early period of an attack of asthma, and cannot, as a rule, be detected after a day or two.

THE EXAMINATION OF THE LARYNX.

LARYNGOSCOPY.

By THOMAS HARRIS, M.D. (Lond.), F.R.C.P.

It is a very common experience in the throat department of a hospital to see a student proceed at once to make a laryngoscopic examination without having paid careful attention to the history of the case, without having considered the general appearance of the patient, and without having examined the neck, the interior of the mouth, or the fauces. He makes, in fact, a series of mistakes, one of the most important of which is that he does what every specialist should carefully guard and watch against doing, viz., becoming a specialist without having first allowed the general condition of the patient to occupy his attention. In the second place, by omitting to consider carefully the history and the symptoms of the case, he runs a great risk of wrongly interpreting any morbid appearances which he may find—in other words, of making a wrong diagnosis with all its attendant evils. And thirdly, he proceeds to make the most difficult part of his examination at once, and is apt to forget to make the easy examination of the neck, of the interior of the mouth, and of the fauces. By such a procedure he is prone to omit the examination of parts which are open to direct and easy observation, parts where lesions may be found which may be of the greatest importance in making a correct diagnosis of morbid laryngeal appearances.

Too much stress can scarcely be laid upon the above points, because such a procedure is so apt to lead to a wrong conclusion. The appearances presented by the larynx in different diseases may be very similar: so close may be the resemblance, that, by considering the laryngoscopic appearance alone, we may be unable to diagnose between several diseases. Conditions may be present elsewhere than in the larynx, the recognition of which may very materially aid us in diagnosing the laryngeal lesion. I have seen a student proceed at once to make a laryngoscopic examination without having first paid any attention to the fauces, which have been the chief seat of the disease, and presented unmistakable evidence of syphilis. The discovery of a syphilitic lesion on the fauces or tongue may help us very much in the diagnosis of a laryngeal condition. The consideration also of a history of the previous existence of cough, with possibly an attack of hæmoptysis having occurred before the symptoms of the laryngeal lesion for which the

patient happens to consult us, may be of the utmost importance. Because, although laryngeal phthisis is in the advanced stage, as a rule, easily diagnosed, in the early stage it is often difficult of recognition. A history of symptoms of phthisis would make us attach much more importance to certain changes in the larynx than we should feel justified in attaching to them if they occurred in an otherwise healthy person who presented only the symptoms and signs of laryngeal disease.

We should first obtain from our patient a short history of his troubles, which will guide us in our future examination of the case. Whilst obtaining this, we note the character of the voice, whether it is natural, husky, or in any way altered, and also the presence or absence of dyspnœa.

In the second place, we take a general view of the patient, and then direct our attention more especially to the nostrils, to the orifice of the mouth, and to the condition of the neck.

Thirdly, we proceed to examine the interior of the mouth and the fauces.

Lastly, we make our laryngoscopic examination.

When obtaining the history of the illness from the patient, we notice whether there is any

Alteration of the Voice.—The voice is, as a rule, more or less altered in laryngeal lesions, but the degree of the alteration bears no relation to the severity of the laryngeal affection, as we may have a very marked and serious affection of the larynx without any appreciable alteration of the speaking voice. It follows from this that the fact of the voice being unaltered is no reason for not examining the larynx, where the detection of a laryngeal lesion might help in the elucidation of the case. Thus cases of paralysis of one vocal cord, from pressure upon a recurrent laryngeal nerve by an aneurysm, &c., are commonly associated with marked alteration of the voice, but complete unilateral recurrent laryngeal paralysis may exist with a natural speaking voice. Also in the serious affection of paralysis of the abductors of the vocal cords, the voice is commonly but little affected. Furthermore, there may be very extensive disease in the larynx at its upper part, where it does not involve the true vocal cords, and yet the voice may not present any marked alterations in character.

These instances will suffice to show how very misleading the voice is as an indication of the presence or absence of a laryngeal lesion. Wherever a complete clinical examination is called for, the fact of the voice being unaltered ought never to be a plea that it is therefore not necessary to make a laryngoscopic examination.

Dyspnœa and Laryngeal Stridor.—At the same time as we

note the character of the voice, we also have an opportunity of noticing the presence or absence of dyspnœa. Dyspnœa of laryngeal origin depends upon the obstruction to the passage of air through the larynx, but the degree of laryngeal stenosis necessary to produce dyspnœa varies very considerably in different individuals, apart from the size of the larynx and the cause of the stenosis. It is remarkable how great a degree of stenosis may exist in some people without causing very great difficulty in breathing. In many cases, before proceeding to a laryngoscopic examination, we may form an idea as to whether the dyspnœa which is present is or is not of laryngeal origin. Laryngeal dyspnœa is commonly noisy, being accompanied by more or less stridor, especially during inspiration. As a rule, it is also associated with an alteration of the voice. There are, however, some important exceptions to both these statements. Thus we may get very marked stridor, closely resembling that due to a laryngeal lesion, in cases of obstruction to the trachea, as from the pressure of an intrathoracic aneurysm or a mediastinal tumour, &c.

The resemblance between the stridor due to laryngeal obstruction and that due to obstruction to the trachea even low down, may be so close that, apart from a laryngoscopic examination, it is impossible to say what is the immediate cause of the dyspnœa. In cases of laryngeal obstruction, the larynx, as a rule, makes a much greater descent during inspiration than is the case where the trachea is the seat of the obstruction. The larynx, however, even in healthy individuals, varies so much in its excursions during deep respirations, and there are so many exceptions to the above rule, that the rule itself is not one to be relied upon. The fallacy of relying upon an alteration in the voice as an indication of dyspnœa being of laryngeal origin has already been called attention to in the case of paralysis of the abductors of the vocal cords, where we may have marked dyspnœa without alteration of the voice.

Cough.—In obtaining the history of the case, we may also have our attention directed to the presence of a cough. The character of the cough in diseases of the larynx varies considerably. In some cases its character is suggestive of a particular laryngeal lesion. The term "croupy" cough is commonly applied to that form of cough which is sometimes heard in membranous inflammation of the larynx, but it may also be frequently heard in children who have a *simple*, non-membranous laryngitis. The peculiar metallic quality of the cough heard in some cases of paralysis of one vocal cord—and, from the most frequent cause of such a paralysis, termed sometimes the "aneurysmal cough"—is also worthy of note. It is, however, to be remembered that in many cases of paralysis of a vocal cord, the cough, if present, has no special characters, and also that a cough very similar to that

above mentioned may be present in some cases where no laryngeal paralysis of any kind exists.

Another important consideration in connection with the existence of a cough in cases of slight lesions of the larynx, pharynx and fauces is the necessity of being quite certain that the cough is really due to the changes in the throat, and not to a more serious, though possibly early, affection of the lungs. This warning appears specially necessary because we so frequently see cases of phthisis where the cough, and often also hæmoptysis, has been put down to quite insignificant changes in the pharynx or larynx, whilst the much more serious pulmonary tuberculosis has been overlooked and valuable time lost.

Before proceeding to describe the method of examination of the larynx, it is necessary to say a few words on the means of illumination,

FIG. 87.—Laryngoscopic Lamp and Bracket. The head-piece has a universal movement round the incandescent gaslight.

and of the instruments which are necessary for making the laryngoscopic examination.

Methods of Illumination.—We may use various forms of light for the examination of the throat. The best light for all purposes is sunlight, but unfortunately it is too often not available for our use. When, however, it can be employed, it should be preferred to any other form of light, as it is not only an intense light, but a pure white one. In the summer months especially it may frequently be employed by placing the patient with his back to a window having a suitable aspect, and reflecting the rays of sunlight from the forehead mirror, just as when we are employing gas or other artificial light. In such cases care must be taken that the sun's rays are not brought to a focus direct upon the uvula or soft palate, as such a procedure would naturally result in injury to those parts.

Gaslight is the illumination which we most frequently employ in towns for throat examinations. A very good light can be obtained from an ordinary Argand burner, a better from an incandescent gas-light, but a more satisfactory one still is derived from either of these in front of which is placed a bull's-eye glass condenser. There are many forms of such lamps in the market; the one represented in the preceding illustration (Fig. 87) will be found a very convenient one, as it not only gives a good white light, being fitted with an incandescent gaslight (Welsbach), but the head-piece has a universal movement capable of revolving round the light on both a horizontal and vertical axis, so that the rays can be more easily directed than in many throat-lamps, which are usually not provided with both these movements.

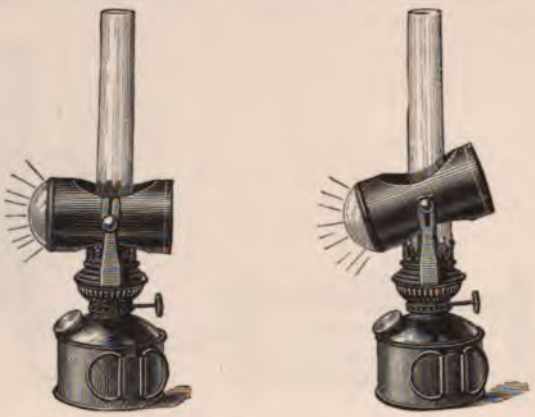


FIG. 88.—Portable Oil Lamp.
A, in position for laryngoscopic examination.
B, in position for microscopic work.

In the country, where gas is not available, and for bedside work in the town, an oil lamp has to be employed. A small portable lamp for this purpose, which is also a very good lamp for microscopical work, is represented in the above illustrations (Fig. 88).

For those who have much throat-work to do, an oxy-hydrogen light will be found of great service, or, where an electrical installation is available, a focal electric lamp with a bull's-eye condenser will be found most satisfactory.

Position of the Patient and of the Examiner.—Whichever form of light is used, the patient should be so placed that the light is close by the side of the head, about on a level with the mouth. It is not a matter of great importance as to which side of the patient the light is placed. Some prefer the light on one side, others always work

with it on the other. It appears, however, somewhat more advantageous to have the light on the patient's right-hand side, because we are then able to make any application to the throat, or perform any manipulation with the right hand, without placing that hand in the rays of light, and so obscuring the view of the throat (Fig. 89).

Reflectors and Laryngeal Mirrors.—Forehead reflectors can be obtained mounted on a spectacle frame, on an ordinary stuff head-band, or better on a steel spring head-piece. Various forms are shown in the accompanying illustrations (Fig. 90 A, B, C, D).

The reflector, whichever form is chosen, should be arranged over that eye of the observer which is nearest to the light; thus, if the light is on the patient's right side, the laryngeal reflector should be placed over the observer's left eye. The reflector should also be so placed that when the light is directed into the patient's mouth, the observer has a clear

FIG. 89.—Position of Patient, Examiner and Lamp when making a Laryngoscopic Examination.

view through the hole in its centre, and can examine the throat with both eyes.

Attention should first be given to the examination of the neck, and the external parts around the orifice of the mouth and the nostrils. We note in doing so whether the larynx is symmetrically placed, whether there are any enlarged glands in the neck or old scars, and whether there is any tenderness of the various regions on palpation. After so much of the examination has been made, the patient is directed to open the mouth, and the interior of that cavity and the fauces are inspected. If the tongue is at all in the way, one of the laryngeal mirrors will be found to make a convenient spatula to place upon the tongue whilst the fauces are examined. The importance of carefully examining the mucous membrane of the mouth and the fauces, before

proceeding with the laryngoscopic examination, has been already sufficiently pointed out. After the above examination has been completed, we proceed to examine the larynx. For this purpose we require a

FIG. 90 A.—Laryngeal Reflector mounted on a Spectacle Frame.

FIG. 90 B.—Laryngeal Reflector mounted on Stuff Head-band.

FIG. 90 C.—Laryngeal Reflector with Steel Spring Headpiece.

FIG. 90 D.—Laryngeal Reflector with Steel Headpiece when folded up.

FIG. 90 E.—The Different Sizes of Laryngeal Mirrors which can be obtained.

FIG. 90 F.—Laryngeal Mirror fitted in its Handle.

small mirror, known as a laryngeal mirror, using a size proportionate to the age of our patient and the size of the throat. These laryngeal mirrors (Fig. 90 E and F) can be obtained of various sizes (Fig. 90 E), and are supplied either fixed in handles, or so arranged that a dif-

ferent-sized mirror can be fitted readily into one handle (Fig. 90 F). In order thoroughly to examine a larynx, it is usually necessary for the patient to protrude the tongue, which is then carefully held outside by the examiner or by the patient, a small napkin, towel, or handkerchief being used for this purpose.

Method of Examination.—The patient protrudes the tongue as far as possible, and a small napkin or handkerchief is then folded round

FIG. 91.—(*After Eichhorst.*)

FIG. 92.—(*After Eichhorst.*)

its tip, and the organ held either by the patient or by the examiner between the thumb and first two fingers of the left hand (Fig. 89). In doing this it is necessary for the beginner to be warned against squeezing the tongue too tightly, and especially against pulling it too vigorously forward. If it is pulled firmly forward, a good deal of pain is caused, and the under surface is readily wounded against the sharp lower incisor teeth.

FIG. 93.—Paralysis of Crico-Arytænoidei Postici. Position of Vocal Cords during Inspiration. (*After Eichhorst.*)

FIG. 94.—Healthy Laryngeal Image during Phonation. (*After Eichhorst.*)

The handle of the laryngeal mirror should be held between the thumb and first two fingers of the right hand exactly as a pen is held (Fig. 89). It is then warmed either by holding it over the lamp, taking care not to heat it sufficiently to damage the mercurial coating, or by dipping it into hot water. Before passing it into the mouth, it must always be tested on the back of the examiner's hand to see that it is not too hot. We next pass the mirror along the dorsum of the tongue, the silvered

face of the mirror being held parallel to its dorsum, and not at right angles to it, until the soft palate is reached, when the mirror is turned so that the reflecting surface is directed towards the examiner, and the uvula displaced slightly upwards and backwards, sufficiently for the interior of the larynx to become visible (Fig. 91). The posterior pharyngeal wall should not be touched by the mirror, if it can be avoided, as such a procedure often occasions retching, and renders the examination of the larynx more difficult.

The beginner in many cases will only see the base of the tongue, the epiglottis and the horns of the larynx (Fig. 95), and will find it probably necessary to alter the angle at which the laryngeal mirror is held

FIG. 95.—(*After Eichhorst.*)

FIG. 96.—(*After Eichhorst.*) Position of parts in the Laryngoscopic Image. *r*=right, *l*=left.

before he is able to obtain a view of the interior of the larynx. Supposing, however, a view of the larynx is obtained, the various parts, commencing from above with the epiglottis, should be observed in a systematic manner. After the parts have been examined whilst at rest (Fig. 91), the patient should be directed to take a deep inspiration, and whilst this is being done the observer should notice whether the cords become slightly more separated, as they ought to do in health (Fig. 92), or whether they tend to become more approximated, as happens when there is paralysis of the abductors (Fig. 93). Afterwards the patient should be directed to phonate, and the best plan is for him to attempt to sing a high-pitched "E." By that means the observer not only obtains a more complete and perfect view of the

larynx, but is able to see whether both cords move perfectly, and whether they come into apposition along their whole length (Fig. 94).

As will be understood from the laws of reflection, the laryngeal image as seen in the mirror is at first somewhat confusing. Whilst objects on the right and left in the larynx are seen in the mirror on the patient's right and left respectively, and appear, therefore, unaltered in position, those parts situated anteriorly, such as the epiglottis, are seen at the upper part of the mirror, whilst those parts most posteriorly situated appear at the lower part of the mirror (Fig. 96). A very short experience, however, of laryngoscopic work is sufficient to accustom the student to the relation of the parts as they are seen in the mirror.

Immediately the examination is completed, the throat mirror should be thoroughly washed in some disinfectant solution, 1 in 20 carbolic acid being suitable for this purpose, so as to avoid any risk of infecting another patient with syphilis, tuberculosis, or other contagious disease. A separate handkerchief or mouth-napkin must, of course, be used for each patient, or a good plan is to use small pieces of linen or cheap cotton material, which can be subsequently burnt.

Some of the Difficulties connected with Laryngoscopy.—The beginner must be prepared to spend a considerable time at laryngoscopy before he is able to see the larynx in the majority of cases which he examines. In some cases it is a very easy and simple matter to obtain at once a perfect view of the interior of the larynx, whilst in other cases the most expert observer may have considerable difficulty in obtaining a satisfactory view of the parts. There are different causes which contribute in different cases to this difficulty.

Some people have throats which are so irritable that it is scarcely possible for them to allow the observer to place the throat mirror in position before they commence to retch. When a difficulty in the examination arises from this cause, it is advisable to withdraw the mirror from the mouth and to wait a few moments before reintroducing it. The difficulty may also be overcome by directing the patient to take a series of very short respirations, to pant, whilst the examination is being proceeded with. Often such a plan will be successful if the mirror is not kept too long in the throat at each examination. If this fails, the local application of cocaine, either in the form of a lozenge,[1] or as a 10 per cent. solution applied by means of a brush to the throat, is the best way of overcoming the difficulty. With children it may be quite impossible, even after the application of a solution of cocaine to the throat, to prevent the retching, and in such cases we

[1] It is well to bear in mind that the majority of cocaine lozenges in the market have no anæsthetising effect; the cocaine appears to be frequently, in some way, destroyed in the preparation of the compressed tablets now so generally in use.

have to persist with the examination despite the retching, and to try to obtain a glimpse of the larynx between the successive spasmodic contractions of the throat.

One of the most frequent impediments to obtaining a perfect view of the larynx is the position of the epiglottis, which not unfrequently lies nearly flat over the orifice of the larynx, and renders it impossible to see more than its horns (see Fig. 95). In some of the cases where the epiglottis is not lying extremely horizontal, this difficulty may be overcome by placing the throat mirror as far back in the pharynx as possible, and altering the angle at which the mirror is held. Frequently also the attempt of the patient to sing a high-pitched "E" is very effectual in giving a view of parts which are not otherwise to be seen. The protrusion of the tongue as far forwards as possible by the patient also assists the object in view. If these plans fail, as in many

FIG. 97.—The various Names attached to the Different Parts of the Laryngoscopic Image. *L*, tongue; *E*, epiglottis; *P*, pharynx; *V*, valleculla; *R*, glottis; *L.v.*, true vocal cords; *L.v.s.*, false vocal cords; *S.M.*, sinus Morgagni; *S*, cartilage of Santorini; *W*, of Wrisberg; *S.p.*, sinus piriformis.

cases where the epiglottis is very horizontally placed they will do, then it is advisable to paint the fauces and the epiglottis with a solution of cocaine, and to mechanically elevate the epiglottis. A very good elevator, and one which is generally available, is a stiff gum elastic catheter. Whilst the patient holds his own tongue, the throat mirror is held in position by the observer with the left hand, the catheter is held in the right and passed into the throat, and over the upper border and posterior surface of the depressed epiglottis, which is then gently raised until a view of the larynx can be obtained. Instead of a catheter a laryngeal sound makes a very good epiglottis elevator.

An enlarged uvula not unfrequently presents some impediment to obtaining a good view of the larynx, and is best overcome by the employment of the largest-sized throat mirror. Enlarged tonsils, on the other hand, necessitate the employment of a small mirror.

The principal object of the present chapter is to teach the method

which it is advisable to pursue in making an examination of the throat and larynx, and especially to show the importance to the beginner of proceeding with such an examination in a careful and systematic manner. It would be beyond the scope and intention of the article if any attempt were made to describe in detail the appearances met with in the various affections of the throat and larynx. It is advisable, however, to consider briefly certain morbid appearances, but more with

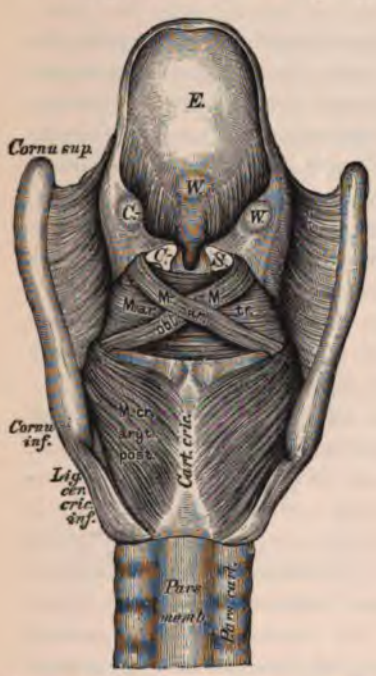


FIG. 98.—Larynx with its Muscles, from behind. *E*, epiglottis with the cushion (*W*); *C.W.*, cart. Wrisbergii; *C.S.*, cart. Santorini; *M.ar.tr.*, musc. arytænoideus transversus; *MM.ar.obl.*, musc. arytænoidei obliqui. (*Landois and Stirling.*)

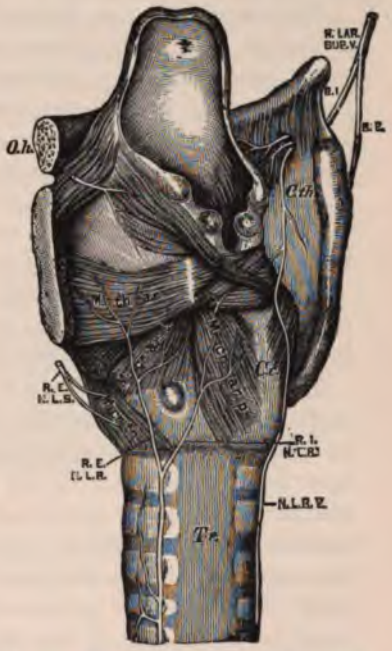


FIG. 99.—Muscles and Nerves of the Larynx. *O.h.*, os hyoideum; *M.th.ar.*, musc. thyreo-arytænoideus; *M.cr.ar.p.*, musc. crico-arytænoideus posticus; *M.cr.ar.l.*, musc. crico-arytænoideus lateralis; *M. cr. th.*, musc. crico-thyroideus; *N. lar. rec. v.*, nerv. laryngeus recurrens. (*Landois and Stirling.*)

a view of still further impressing upon the student the necessity of educating himself to observe the different parts of the throat and larynx in a systematic manner, than to try to teach the appearances which enable us to diagnose any particular case.

Normal Laryngoscopic Appearances.—The various names attached to the different parts of the larynx, as seen in the laryngeal mirror, will be better learnt by a consideration of the foregoing diagram (Fig. 97) than by any lengthy description.

The two illustrations (Figs. 98 and 99) show most of the important muscles of the larynx, and will serve to refresh the memory of the student who is not engaged in anatomical and physiological work.

Morbid Conditions of the Larynx.—After having obtained the history from the patient, and noticed his general appearance, the condition of the neck, and the interior of the mouth and fauces, we proceed with the examination of the larynx in the manner above described.

Our attention is first directed to the colour of the parts presented to view in the throat mirror, and we note the colour of the various parts, proceeding in a systematic manner from above downwards, first observing the epiglottis, then the ary-epiglottidæan folds and the horns of the larynx, then the external walls of the larynx, including the false cords or ventricular bands, the posterior wall of the larynx, the true vocal cords, and lastly so much of the trachea as is possible in the particular case. In some instances we may be able to see the bifurcation of the trachea and the orifices of the main bronchi.

Colour.—We note the colour of the various parts, and remember that the colour of the mucous membrane of the larynx varies not only in different regions of the organ, but in different individuals.

The true vocal cords, as a rule, stand out prominently and contrast markedly in their colour with other parts of the larynx. They are, as a rule, pale, almost perfectly white, but in some individuals the contrast between the colour of the true cords and that of other parts of the larynx is not so great, and the cords may present a considerable amount of redness, without being in a pathological condition, without warranting us in describing the laryngeal mucous membrane as hyperæmic, or in a state of inflammation. An unusual pallor, an anæmic condition of all parts of the larynx, may be found in cases of general anæmia, but we must remember that a pallor of the larynx is very common in the early stages of tuberculosis of the organ, and that this is commonly associated with a paleness of the mucous membrane of the soft palate and pharynx, and with a prominence of the small venules on the otherwise anæmic parts of the fauces.

A morbid redness is seen in simple congestion or in inflammation of the larynx. On the surface of the mucous membrane of the larynx we may find different forms of secretion. One of the most common is a simple mucoid or muco-purulent secretion, which is often deeply charged and coloured black with inhaled carbonaceous matter, an appearance frequently seen in simple laryngeal catarrh. In other cases a more purulent secretion is visible, and in rarer cases a fibrinous or necrotic membrane, as in membraneous laryngitis, or as in diphtheria of the larynx.

Thickening and Tumours connected with the Larynx.—From a consideration of the colour of the mucous membrane we pass to notice any thickening of the different parts, again proceeding in our observation in a systematic manner from above downwards, and noticing each part in turn. It is in connection with thickening of the mucous membrane that the greatest variety is met with in laryngeal diseases, and the greatest difficulties are found in interpreting the appearances. We note not only the seat of thickening, but whether it is a localised one or passes gradually into the surrounding mucous membrane. The most typical localised swellings are seen in the benign growths, the simple polypi, which project to a greater or less degree into the interior of the larynx. These are commonly multiple, and are most frequently found to spring from the true cords (Figs. 100, 101, *A.* and *B.*). It is to be remembered, however, that occasionally single and localised growths are not benign, but that malignant disease in its early stage may take

FIG. 100.—Solitary Papilloma growing from the left true vocal cord. (*After Burnett.*)

A. *B.*

FIG. 101.
A. Solitary Laryngeal Polypus springing from the right vocal cord.
B. Ditto, during phonation.
(*After Lennox Browne.*)

that form. We have also to be careful not to mistake the edge of an ulcer for a new growth, a mistake which can be readily made. It is not very uncommon to meet with cases where even experienced laryngologists have a difficulty in determining whether an apparently localised swelling is an actual new growth or only the upper edge of an ulcer. This difficulty will be readily appreciated when it is understood that it is often impossible by any means to obtain a view of the base or floor of a laryngeal ulcer. Often the only part of an ulcer which can be seen is its upper edge, and if this is very much thickened and stands out prominently into the interior of the larynx, it may have a very close resemblance to a new growth. This deceptive appearance is probably nowhere in the larynx so frequently met with as on the posterior wall of that organ, and in that position we have constantly to be on our guard against mistaking the edge of an ulcer or a simple inflammatory thickening for a true new growth.

The posterior wall of the larynx is a not uncommon seat of ulcerations or inflammatory thickenings, whilst it is a much rarer position in which to find any form of new growth arising.

In connection with any local thickening or apparent new growth in the larynx, it is important to also note whether the vocal cords move perfectly on phonation. Cases of primary malignant disease usually, but not invariably, are associated at even an early state of their development with a fixing of one or other of the vocal cords.

Ulcers of the Larynx.—After observing the colour of the mucous membrane of the larynx and the existence or non-existence of any thickening, we note the presence or absence of any ulcerations.

In the larynx a very great variety of ulcerations occurs, and it would take too much space and be otherwise undesirable to give all the points which are of importance in diagnosing one form of laryngeal ulcer from another. It may be stated, however, that it is necessary to notice whether we have one or more ulcers, the position of such, the character and colour both of their surfaces and their edges, and whether they are or are not associated with much surrounding infiltration of the mucous and sub-mucous tissues, and whether such infiltration presents a pale or hyperæmic appearance; and lastly, whether the true cords do or do not move during phonation.

All these points may be of importance in coming to a conclusion as to the cause of the ulceration; for example, whether it is tubercular, syphilitic, or malignant.

Tubercular ulcers in the larynx present a great variety in their appearance, and some of the most difficult laryngeal cases which we meet with for diagnosis are those where it is a question of a lesion being tubercular or otherwise. Usually the tubercular laryngeal ulcer occurs in a multiple form, several existing in different parts of the larynx. Each ulcer is small and of a greyish appearance, and may be seated anywhere in the larynx, but the most common positions are the anterior aspects of the posterior wall, the processus vocales, the posterior extremities of the true cords, and the ventricular bands. Tubercular ulcers are usually associated with a marked pallor of the laryngeal mucous membrane, and frequently by considerable infiltration of their surrounding parts.

The advanced cases of tubercular larynx with the swollen pale ary-epiglottidæan folds, on which are numerous small greyish superficial ulcerations, scarcely admit of being mistaken for any other affection (see Figs. 102, 103, 104). It is, however, in the earlier stages of tubercular disease of the larynx that difficulties so commonly present themselves, and in those cases the greatest attention should be paid not only to the appearances of the laryngeal lesion and the parts of the

larynx, but to the previous history, family history, general appearance of the patient, and the condition of the other organs of the body, especially of the lungs.

It is always best to err on the safe side, to give a guarded prognosis and opinion of a laryngeal lesion where the local appearances are not conclusive of it being tubercular, but where the family history, previous

FIG. 102.—Tubercular Larynx. Thickening of the Epiglottis and Ary-Epiglottidæan Folds, the right side being more affected than the left. (*After Burnett.*)

FIG. 103.—Tubercular Larynx. Similar to, but more advanced than, that shown in Fig. 102. (*After Burnett.*)

history, or present state of the patient are such as to warrant a belief that the laryngeal lesion may be tubercular.

Syphilitic ulcers in the larynx, except the early lesions appearing as superficial erosions or mucous plaques, are frequently solitary, are of considerable size, usually much larger than the tubercular, and cause considerable destruction of tissue. They may be seated anywhere, attacking most frequently the epiglottis. They are commonly associated

FIG. 104.—Extensive Tubercular Ulceration of the Larynx. (*After Lennox Browne.*)

with much redness rather than with pallor in their immediate neighbourhood.

In considering the possibility of any laryngeal ulcer being malignant or otherwise, it is to be remembered that usually in carcinoma of the larynx, it is not in the early stage of the disease that the ulceration chiefly attracts our attention, so much as the thickening and swelling of the part attacked by the disease. The frequency with which, even in an early stage, carcinoma of the larynx causes a fixing of one or

other vocal cord has already been referred to, and is a point of importance in the diagnosis.

Some Laryngeal Paralyses.—After we have noted the colour of the laryngeal mucous membrane, the presence or absence of any swellings or ulcerations, it is a good plan to tell the patient to take a deep breath, and to note whether the vocal cords become more widely separated, as they should do in health (see Fig. 92), from the action of the

FIG. 105.

FIG. 106.

Paralysis of the Left Recurrent Laryngeal Nerve. Laryngoscopic image during inspiration (Fig. 105), and during phonation (Fig. 106). (*After Eichhorst.*)

posterior crico-arytænoid muscles. When those muscles are paralysed, the cords are even at rest more closely approximated than in health, and instead of their separating on a full inspiration being taken, they tend to come nearer together (see Fig. 93), and hence to produce both laryngeal stridor and dyspnœa.

After taking a deep inspiration, the patient is told to say "E," and we notice whether both cords move and become perfectly approximated

FIG. 107.—Paralysis of both Thyro-arytænoidei Interni. (*After Eichhorst.*)

FIG. 108.—Paralysis of both Arytænoidei Postici. (*After Eichhorst.*)

in their whole length. We may find that one cord is fixed, does not move on phonation, but that the other cord crosses over the median line and comes more or less perfectly in contact with it. This is seen in paralysis of one recurrent laryngeal nerve (see Figs. 105 and 106). Or the cords on phonation may leave an oval space between them, as seen in paralysis of both thyro-arytænoidei interni (see Fig. 107), or

whilst the cords come into contact in the greater part of their length, the posterior third of the glottis remains as a triangular opening during phonation (see Fig. 108), as in paralysis of the arytænoideus muscle. We may get a combination of the two latter paralyses, giving the appearance seen in Fig. 109.

FIG. 109.—Paralysis of both Thyro-arytænoidei Interni and Arytænoidei Postici. (*After Eichhorst.*)

FIG. 110.—The Trachea and the Openings of the Main Bronchi. (*After Eichhorst.*)

After observing the movement of the vocal cords, we conclude our examination by noticing the condition of so much of the **interior of the trachea** as is visible. The extent of the trachea which can be seen varies in different individuals. In favourable though somewhat rare cases, we may see the bifurcation of the trachea and the openings of the main bronchi (Fig. 110).

CHAPTER VII.

EXAMINATION OF THE CIRCULATORY SYSTEM.

IN studying diseases of the circulatory system, we cannot fail to be struck with the inconstancy of any definite relation between symptoms and physical signs. The presence of certain physical signs enables us to assert the existence of heart disease, but furnishes us with comparatively little information as to the gravity of the complaint—as to the effect of a particular lesion on the condition of the patient. Three classes of cases illustrating the varying degree in which symptoms and physical signs are correlated may be distinguished.

I. Cases where the physical signs of disease of the heart or arteries are unmistakable while general symptoms are entirely absent. Examples: (1.) An applicant for insurance has had good health and feels quite well, but, on listening to his chest, a loud diastolic murmur is

heard over the sternum, and the apex beat is a little stronger than natural. There is certainly aortic regurgitation, but the leakage is probably slight, and, at any rate, is fully compensated by the slight degree of hypertrophy of the left ventricle. (2.) A gouty man of forty is examined, and the rigidity and tortuosity of his brachial and radial arteries afford distinct evidence of arterial degeneration. He feels and appears to be quite well, but, although young in years, his hold of life must be regarded as precarious. Perhaps one day a degenerated cerebral artery ruptures, and death speedily results from cerebral hæmorrhage.

II. Cases in which physical signs and other objective manifestations of cardiac disease, as dropsy, are associated with various subjective phenomena, as pain, palpitation, shortness of breath, &c. Example: Disease of the mitral valve with insufficient compensation.

III. Cases in which general symptoms are dominant, physical signs of cardiac disease being slight or absent. Examples: (1.) Septic endocarditis. The symptoms of this disease may closely resemble those of pyæmia or of typhoid fever. There is much constitutional disturbance, pyrexia of remittent or intermittent type, while sooner or later embolic seizures occur. A physical examination of the heart may reveal valvular disease without evidence of cardiac enlargement, or merely a disturbance of the frequency or rhythmical action of the heart. (2.) Angina pectoris. This disease is characterised by paroxysmal attacks of intense pain in the chest, together with a sense of impending death, but these symptoms are not *necessarily* attended by any physical signs of cardiac disease.

The necessity for an adequate consideration of symptoms is particularly manifest in connection with the relative importance of lesions of the valves and muscular tissue of the heart. A slight valvular lesion may give rise to a loud murmur but produce no appreciable effect on the cardiac muscle, whereas a severe valvular lesion will quickly lead to dilatation and hypertrophy of the heart. But as long as the cardiac enlargement is merely compensatory—that is, as long as the muscular hypertrophy is able to cope with the valve defect—there need not be any decided circulatory disturbance. Circulatory disturbance, however, soon becomes prominent when compensation fails, and its symptoms give us far more valuable information with regard to the condition of the heart than mere physical signs.

Hence, in investigating a case of heart disease, we must endeavour to make out not only what part of the heart is diseased, but what is the condition of the circulation *through* the heart. For a heart may be considerably diseased, and yet, if the circulation through it is properly maintained, the patient's condition, for the time being at least,

is satisfactory, and there will be but few, if any, of the symptoms of cardiac disorder.

It is, then, to symptoms of cardiac failure that we would here draw the student's attention. The cardinal ones are dyspnœa, dropsy and congestion of the liver. These symptoms indicate weakness of cardiac muscle, whether this occurs as a result of a severe valve lesion or independently of such lesion. In the latter case the weakness may be due to failure of muscle power.

Dyspnœa.—One of the first indications of cardiac failure is shortness of breath, which is at first experienced by the patient only after making exertion. In advanced cases dyspnœa becomes constant, and the patient is obliged to sit up in bed and bring into use the extraordinary muscles of respiration. This *orthopnœa* is often associated with great restlessness, especially at night, and the patient is frequently unable to obtain more than a few snatches of sleep. Sometimes the breathing assumes the *Cheyne-Stokes type*, and then symptoms indicating considerable cerebral disturbance are often present.

Dropsy.—The dropsy resulting from heart disease usually begins in the feet and gradually extends upwards. It may involve the serous membranes, giving rise to ascites, hydrothorax and hydro-pericardium. As a rule, the face and eyelids are spared, except towards the end of a case, when it must be admitted that the kidneys are usually, though not always, implicated. But it needs to be borne in mind that cardiac dropsy is often very widespread, and sometimes presents peculiarities as regards localisation. Caprice of localisation may be frequently observed in cases of alcoholic dilatation of the heart. Thus the neck, arms, and upper part of the trunk, or the back and scrotum, may be considerably swollen when the legs are comparatively or even entirely spared.

Congestion of the Liver.—An enlarged tender liver, as a result of passive congestion, is a prominent feature of cardiac failure, whether this be secondary to valvular disease or occur primarily in consequence of muscle failure. In some cases of alcoholic dilatation of the heart, hepatic enlargement may partly depend on interstitial hepatitis, but that it is commonly due to passive congestion is clearly shown by the rapid diminution in the size of the liver when the circulation becomes restored.

ANATOMICAL RELATIONS OF THE HEART.

Irregularly conical in shape, the heart occupies an oblique position in the chest; the broader end or base is directed upwards, backwards, and to the right, and extends vertically from the level of the fifth to

that of the eighth dorsal vertebra; the apex points downwards, forward, and to the left, to reach the fifth left intercostal space just within the left nipple-line.

The greater portion of the anterior surface of the heart is constituted

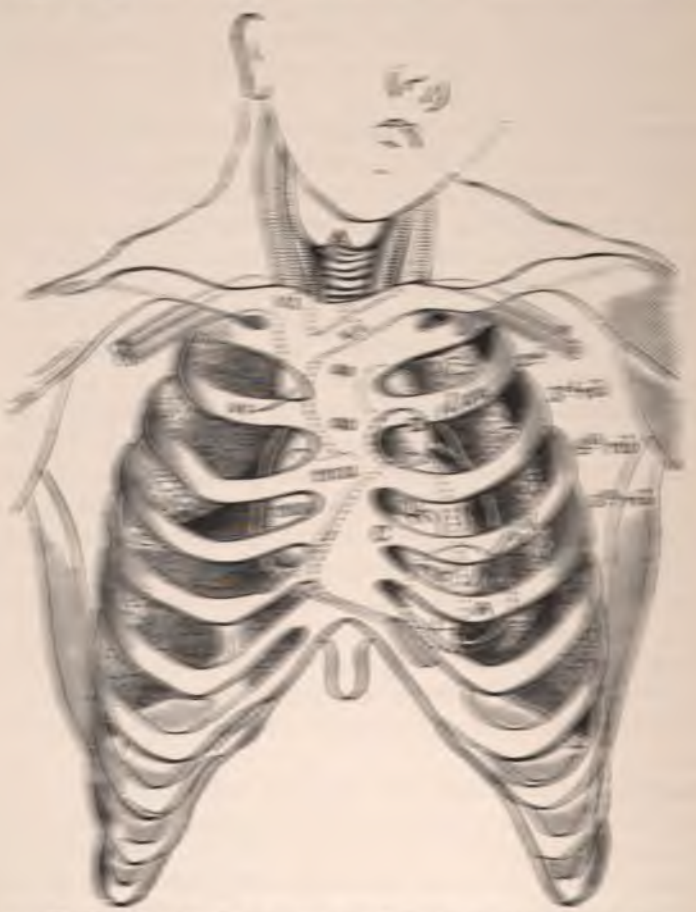


FIG. 111.—The Chambers of the Heart and the Great Vessels in their relations to the Front of the Chest. The lungs are enlarged at their anterior margins, so as to meet, covering in the heart; ra., right auricle; rv., right ventricle; la., left auricle; lv., left ventricle; ao., arch of aorta, traced in dotted outline as far downwards as ; pa., the two pulmonary veins. The letters a, c, t, and in the centres of the areas of projection of mitral, tricuspid, and pulmonary valves respectively. (Luschka and Gerdy.)

by the right ventricle, the remaining portion by a narrow strip of the left ventricle, by the right auricle, and by the two auricular appendices, the right appendix being lower and more anterior than the left. The right border, about half an inch to the right of the sternum, is formed almost entirely by the right auricle, the lower by the right ventricle,

and the left border by the left ventricle. The highest portion of the heart is formed by the left auricular appendix, which lies in the second intercostal space, close to the left margin of the sternum. The lowest portion corresponds to the sixth costal cartilage.

The portion of heart uncovered by lung is shown in Fig. 82, and the line "c, d," which marks the boundary of the complemental pleural space on the left side, indicates the extent to which the superficial cardiac area may be diminished in size during a deep inspiration.

In children, the heart, as well as the diaphragm and the lower edges of the lungs, stand about a rib higher than in the adult. The heart, too, is relatively larger in the child, and has a wider connection with the anterior chest-wall.

In old age, the heart occupies a lower and more horizontal position than in the adult. This is partly due to cardiac enlargement, and partly to emphysema.

As regards the two large **arteries** which spring from the base of the heart, it is to be remembered—

(1.) That the pulmonary artery approaches the surface most closely at the third left cartilage, while the aorta is most superficial at the second right cartilage close to the sternum. As sounds generated at the orifices of these arteries are best heard at the cartilages named, the second right has been named the *aortic cartilage*, the third left the *pulmonary cartilage*.

(2.) That the transverse portion of the aortic arch commences at the junction of the upper edge of the second right cartilage with the sternum, and crossing behind the lower half of the manubrium, passes downwards and backwards, to end in the descending portion of the aortic arch at the lower border of the left side of the fourth dorsal vertebra.

(3.) That the descending portion of the arch of the aorta is in contact with the left side of the body of the fifth dorsal vertebra, and at the lower end of this vertebra terminates in the descending thoracic aorta, which itself ends in the abdominal aorta at the level of the twelfth dorsal vertebra.

INSPECTION AND PALPATION.

The chief points to be observed are :—(1.) The condition of the veins at the root of the neck and over the surface of the chest. (2.) The condition of the large arteries, especially of the carotids, subclavians, brachials and radials. (3.) The condition of the chest-wall in respect to undue prominence and pulsation of the parts in front of the heart and its great vessels.

Venous Distension.—The degree of visibility of the jugular veins varies considerably in different healthy individuals, especially with regard to the stoutness or leanness of the body. In the former condition they may be quite invisible, in the latter their form and colour may stand out distinctly. Distension of the jugulars indicates obstruction to the flow of blood into the right ventricle or in some part of the pulmonary circuit. Thus it is a marked feature in cases of dilatation of the right ventricle, as a result of bronchitis and emphysema or of mitral disease, and in cases in which the superior vena cava is compressed by a mediastinal tumour. In congenital cyanosis jugular distension often reaches an extreme degree.

Associated with fulness of the veins at the root of the neck there is often distension of the veins of the upper part of the chest, as well as of those of the head, neck and arms. When there is much obstruction to the return of blood to the heart, the venous distension of the upper part of the body becomes extreme, and is associated with œdema. In such cases the face, neck, chest and arms are greatly swollen, and present a dark purplish appearance. The condition is a more common result of a solid intra-thoracic growth than of an aneurysmal tumour. In connection with undue prominence of superficial veins on the surface of the chest, it may be mentioned that a band of purplish venules is not uncommonly seen across the lower part of the thorax quite apart from disease. Its most usual position is a little above the costal margin; it extends as a continuous band from one side to the other, crossing the middle line at the level of the xiphoid cartilage, and is usually better developed on the left than on the right side. Its significance has not been determined, but it is certainly met with in many individuals who appear to be in perfect health.

Venous Pulsation.—Slight pulsations along the jugular veins may be occasionally observed without there being any grave disturbance of health, as in simple anæmia. They are best observed on the right side of the neck. The patient should be recumbent, with his head thrown slightly backwards and to the left. To distinguish venous from arterial pulsation, the finger should be laid very lightly over the lowest part of the distended vein; then, if the pulsation cease, it is obvious that it occurred in the vein itself; whereas if the pulsation continue, it is evidently arterial. Further, if while the finger still compresses the vein, a finger of the other hand be drawn upwards along the vein in order to empty it from below, then, on removing the finger from the root of the neck, the vein, in cases of venous pulsation, will be seen to fill from below, either gradually or quickly, according to the degree of incompetence of the tricuspid orifice, provided that the venous valves are themselves incompetent. In order to time venous pulsation, it

should be observed in relation with the heart's apex beat or with the pulsation of the left carotid. The recent observations of Ringer and Sainsbury, and of Mackenzie have shown the importance of determining not only the time of the venous pulse in relation to that of the cardiac systole, but also the size and duration of the waves produced in the vein by the contractions of the right auricle and ventricle. For the following account the author is indebted to Dr. Mackenzie, who has so ably investigated the bearings of this important subject:[1]—

"Under certain conditions which produce dilatation and engorgement of the right heart, a backward movement of the blood is induced in the veins near the heart. This backward movement is chiefly manifested in the veins of the neck—external and internal jugulars—but it may rarely extend to the superficial veins of the head, trunk and extremities. The appearance of this 'venous pulse,' as it is called, varies very much in different individuals. During one cardiac revolution but one wave may be apparent, and its time of appearance depends on whether it is caused by the systole of the auricle or by that of the ventricle. On the other hand, there may be distinctly perceptible two waves, or several occurring with such rapidity that their appearance resembles dancing vibrations. Yet each of these smaller movements can be distinctly attributed to a definite cause when a graphic record of them is obtained simultaneously with a standard time, such as the arterial pulse, carotid, or radial. This object is attained by means of the clinical polygraph. This instrument consists of two portions, a sphygmograph combined with a phlebograph. The phlebographic part consists of a small lever and tambour, the tambour being connected by india-rubber tubing with a small shallow cup called a 'receiver.' The receiver, laid over any pulsating part in such a manner that communication with the outside is prevented by the close adaptation to the skin, readily conveys the movements to the tambour. By means of a light stem the tambour is fixed to the upright bar that supports the lever of a Dudgeon's or von Jacquet's sphygmograph. By suitable movements the lever of the phlebograph can be made to write on the same paper and at the same time that the sphygmograph inscribes the movements of the radial pulse. In this manner such varied movements as apex beat, carotid pulse, jugular pulse, and liver pulse can be graphically recorded at the same time as the radial pulse on an ordinary-sized sphygmograph paper, as in Fig. 112. With these varied movements thus recorded, the interpretation of the venous pulse becomes a simple matter. It is assumed that the pulse in the jugular veins appears at the same time as that of the carotid, and the difference in time between

[1] Dr. Mackenzie's papers are published in the *Journal of Pathology and Bacteriology*, 1892–94.

the carotid and radial pulses being noted, the events preceding and following it can be attributed to the known movements of the heart. On account of the proximity of the jugular vein to the carotid artery, the tracing of the venous pulse very frequently cannot be obtained quite free from the shock communicated to the instrument by the carotid pulse. This wave is therefore frequently present in the tracing, as in Fig. 112, where the wave (*c*) is due to the carotid impact, and is called the '*arterial wave.*' It is to be noted that it is absent from the liver tracing. Taking the wave (*c*) as the time of the carotid pulse, the wave (*b*) preceding it can only be due to the systole of the auricle; hence it is called the '*auricular wave.*' After its systole the auricle passes into diastole, dilates, and sucks in the blood from the veins. In consequence of this, the hitherto distended vein quickly empties, and this emptying is represented in the tracing by the great fall called the '*auricular depression,*' the line of descent being interrupted by the shock from the carotid artery producing the arterial wave (*c*). A point of great interest is the manner in which this auricular depression is

FIG. 112.—The upper tracings are taken from the apex beat, carotid, jugular and liver pulses, at the same time as the radial pulse (lower tracing). The tracings from the right internal jugular vein and liver show the waves characteristic of the venous pulse and liver pulse of the auricular type; *b*, the auricular wave; *c*, the arterial wave; *a'*, the part of the ventricular wave before, and *a*, the part after the closure of the pulmonary valves.

terminated. This depression being caused by the diastole of the auricle, will cease when the distension of the auricle is completed. In health, when the tricuspid valves are competent, the distension of the auricle will be accomplished by the blood from the veins alone. But if the tricuspid valves are incompetent, then the auricle will fill from blood being poured in from two sources, namely, from the venæ cavæ, and back from the ventricle. Hence it follows that the greater the incompetence of the tricuspid valves, the earlier will the auricle fill, and the sooner will the auricular depression in the venous pulse be terminated. This termination is therefore practically due to the appearance of a new wave, '*the ventricular.*' As this new wave increases in size, the time occupied by the auricular depression diminishes, and in this manner we obtain an idea of the amount of tricuspid incompetence. In Fig. 112 this ventricular wave is represented by *a'* and *a*. The division of this wave into two parts occurs in this wise. The auricle being filled,

and the ventricle still contracting, the blood is poured through the incompetent tricuspid orifice and forced back into the veins, giving rise to the first part of the ventricular wave (the size of the wave being also increased by the stasis in the veins). When the pulmonary valves close, the blood that has hitherto been escaping from the ventricle by the two orifices (pulmonary and tricuspid) is suddenly checked in its escape through one orifice by the closure of the pulmonary valves. At the moment that this happens there will be an increased flow through the other orifice, and it is manifested by an increase in the size of the ventricular wave of the venous pulse. Thus the interruption on the ventricular wave between a' and a is therefore the signal of the time of closure of the pulmonary valves. The diastole of the ventricle succeeding, the blood again pours from the auricle and from the vein, giving rise to the depression between the waves a and b, which is therefore called the ventricular depression. This is followed by the systole of the auricle and the production of the auricular wave, and so the series of events begins to recur.

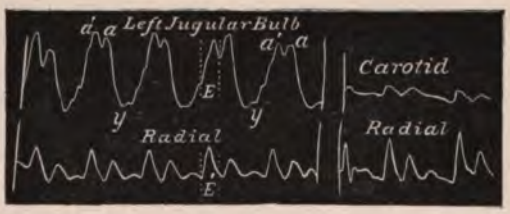


FIG. 113.—Simultaneous Tracings of the Left Jugular Pulse and of the Radial Pulse. The venous pulse is of the ventricular type. The spaces E and E' represent the time occupied by the ventricular outflow in venous and radial pulses.

"The venous pulse thus described is that characteristic of 'functional' dilatation of the heart, as in chlorosis, pregnancy, debility, &c. It may also be due to the dilatation consequent on organic disease of the heart. Inasmuch as the auricle is fairly efficient, and its movements are those chiefly reflected into the veins, this form of pulse is called the 'auricular form' of the venous pulse, to distinguish it from the 'ventricular form.' This latter is merely a more advanced stage than the former, in which the tricuspid incompetence has been so great that the auricle has become over-distended and paralysed, so that it no longer reflects its movements in the veins. In such cases the ventricular wave is the only wave present, and is due to the direct communication between the ventricle and the veins. In such cases we have but one great wave—the ventricular—synchronous with the carotid pulse, as in Fig. 113, with one great fall—the ventricular depression—due to the diastole of the ventricle. On the top of such a wave there is frequently a notch, signi-

nificant of the closure of the pulmonary valves in the manner already explained. Stages intermediate between these two forms and the passage of the auricular into the ventricular can occasionally be demonstrated by continuous observations in individual cases. The liver pulse shows practically the same features, presenting an auricular form of pulse and a ventricular form. The liver pulse never occurs unless there is some organic lesion of the heart, and is never present from mere *functional* dilatation.

"In chronic mediastinitis there is sometimes an additional depression in the pulse due to the sudden diastole of the ventricles permitting a rebound of the costal cartilages which expands the chest and sucks the blood in from the veins.

"Inasmuch as the venous pulse is the outward and visible sign of increased venous pressure, and as there is a distinct relationship between the arterial and venous pressures, the observation of the venous pulse serves as a very delicate guide in detecting variations in the blood pressure in certain cases. The venous pulse also affords instructive information concerning the movements of the different chambers during irregular action of the heart. When a record is taken simultaneously of the apex beat or arterial pulse with the venous pulse, a want of rhythm in the movements of the cardiac cavities can in suitable cases be demonstrated."

Superficial distended veins on the front of the chest may occasionally be seen to pulsate in cases of tricuspid regurgitation.

The Arteries.—In addition to the pulse at the wrist, which will be subsequently considered, some of the other superficial arteries of the body need to be carefully examined, especially as regards the degree of their pulsation and the condition of their walls.

Pulsation of the superficial arteries is but little visible in health, except after great bodily exertion or in conditions of mental excitement. In many morbid conditions, however, it becomes a conspicuous feature. Thus throbbing of the carotids, subclavians and brachials is often seen in anæmia and in Graves's disease. The most extreme pulsation occurs in cases of free aortic regurgitation; it is most characteristically seen in large arteries like the carotids, but small ones like the temporals or facials also show pulsation. Marked arterial pulsation is often conspicuous in cases of enlargement of the left ventricle, and especially when the walls of the arteries have lost their normal elasticity in consequence of degenerative changes.

Great attention should always be given to the **condition of the arterial walls** as revealed by palpation of the temporal, brachial, radial, and other superficial arteries. The brachial is a convenient and important artery to examine in this respect. The arm should

be bared and semiflexed at the elbow. Then any undue prominence, tortuosity, or unusual movement of the vessel may be observed, while the condition of its walls may be investigated by rolling it under the fingers, and by pressing them gently along the course of the artery. There may be uniform or irregular thickening—in the latter case, indurated patches or calcareous plates may be felt, which are sometimes arranged in a moniliform manner. When there is considerable atheromatous degeneration, or when there is aortic regurgitation apart from arterial disease, a distinct "locomotive pulse" is often visible—that is, considerable movement of the brachial artery accompanies each cardiac systole. Rigid arteries stand out like cords, and their tortuosity is accentuated by each contraction of the heart.

The arteries of the two sides of the body should be compared, and any **want of symmetry** carefully investigated. Thus unilateral pulsation in the neighbourhood of the sternal end of the clavicle may be due to an aneurysm of the innominate artery. In such a case the pulsations of the carotid, subclavian and brachial arteries will be feebler on the right than on the left side.

Unsymmetrical pulsation also occurs in aneurysms affecting the transverse portion of the aortic arch, and will be referred to again.

The Precordia, or that portion of the chest-wall which overlies the heart, is formed by the third, fourth and fifth left costal cartilages with their adjoining interspaces and the adjacent piece of the sternum. In health the contour of this region does not differ from that of the corresponding portion of the right front.

Undue Prominence or Bulging of the precordia as a result of heart disease is much commoner and more marked in the child than in the adult. It usually results from cardiac enlargement, rarely from pericardial effusion; when the latter is considerable, the intercostal spaces may reach the level of their cartilages or even protrude beyond them, the integuments then often becoming œdematous.

Depression or Retraction of the Precordia may result, in rare cases, from absorption of fluids effused into the pericardial sac, or possibly in consequence of extensive pericardial adhesions.

The Apex Beat may be defined to be the lowest and outermost point of the heart's impulse which strikes against the chest-wall.

In healthy adults the apex beat is seen and felt in the fifth left interspace, and well within the left nipple-line. Its extent is not greater than a square inch, and may be covered by the end of the thumb. In persons with a short wide thorax the apex beat may stand in the fourth interspace; also in children under ten years of age it is usually found in the fourth space, and either in the nipple-line or possibly a little outside it; in many old people it occupies the sixth

interspace. In fat or very muscular chests the apex beat may be invisible, but it can usually be felt by pressing deeply on the chest while the patient leans forward. In health the apex beat, as above defined, is the only visible cardiac impulse, but neither its absence nor the presence of an epigastric impulse can be regarded as necessary indications of disease.

While quiet breathing does not affect the position of the apex beat, a deep inspiration may depress it half an inch. Lying upon the left

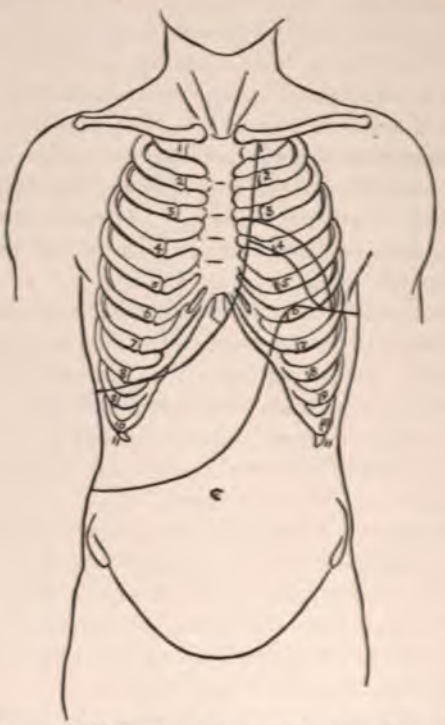


FIG. 114.—Displacement of Mediastinum, Heart and Liver from Pneumothorax of the right side. (*Weil.*)

side displaces the apex beat to the nipple-line or beyond it. When a person lies upon the right side, it may disappear, or be perceptible to the right of its normal position. Bodily exertion or nervous disturbance tends to increase its strength and area, and the throbbings of the right ventricle may be felt in the epigastrium.

Changes in the Position of the Apex Beat as a Result of Disease.—The apex beat is **displaced upwards** whenever the diaphragm is raised, as by abdominal tumours, ascites, or tympanites; in cases of shrinking of the upper portion of the left lung, as from chronic

phthisis; and in pericardial effusion. It is **displaced downwards** when the diaphragm is depressed, as in emphysema or pneumothorax, when it is usually also feeble; in aneurysm of the aortic arch, and in hypertrophy of the left ventricle. It is **displaced to the right** by left pneumothorax or by left pleuritic effusion. In such cases a cardiac impulse, not usually the apex beat, may be perceptible even as far out as the right mammary line. Shrinking of the right lung also displaces the heart to the right of its normal position.

It is **displaced to the left** by fluid in the right pleura, when the apex beat may be felt as far out as the mid-axillary line; and in enlargement of the left ventricle. It is to be noted that effusions into the right pleura displace the heart far less than effusions into the left pleura.

The Strength of the apex beat **is increased** most markedly in hypertrophy of the left ventricle, a condition which commonly results from aortic or Bright's disease; if it be strong enough to push forward the finger, exerting moderate pressure, it is called "heaving." It is also increased in cases of exophthalmic goitre.

Diminution or Absence of the Apex Beat occurs when the heart is unduly covered by lung, as in emphysema, or surrounded by fluid, as in some cases of pericarditis; or when its action is enfeebled in any way, as in fatty degeneration; and sometimes in cases of adherent pericardium.

Extent and Position of the Cardiac Pulsation.—Increase in strength of action is usually accompanied by increase in the area of the apex beat, and then often enough cardiac pulsation is not limited to the apex, but is felt, as in a dilated hypertrophied heart, over the greater part of the precordia. But an extensive diffuse impulse is not necessarily a strong one, and may exist apart from a definite apex beat. Thus in pericardial effusion a weak wavy impulse may be perceptible in the third, fourth and fifth spaces, and it is then often impossible to define the apex beat, or even to say that the impulse has any point of maximum intensity. Also in enlargement of the right ventricle, and when dilatation greatly exceeds hypertrophy of the left ventricle, the true apex beat tends to disappear. Marked and extensive cardiac pulsation occurs when the heart is pushed forwards as by a tumour of the left lung or in the mediastinum.

Epigastric Pulsation is produced by any condition, such as emphysema, which lowers the diaphragm, or which pushes the heart towards the right; it is also a valuable sign of enlargement of the right ventricle, and hence of embarrassed pulmonary circulation. In the last class of cases it is worthy of notice that the pulsation affects the *xiphoid cartilage* and adjacent left cartilages rather than the epigas-

trium: this is a distinction from pulsation of the liver, which is occasionally present when there is great tricuspid regurgitation and is more truly epigastric, and from pulsation of the abdominal aorta.

Pulsation in the Second Left Space is sometimes caused by a distended pulmonary artery or by dilatation of the conus arteriosus, especially the latter.

Pulsation of the Right Auricle is occasionally perceptible to the right of the sternum, and is significant of extreme distension of the right side of the heart.

A Diastolic Shock accompanying closure of the pulmonary valves is sometimes felt in the second or third left space in chronic cases, where the pulmonary circulation is obstructed.

Systolic Depressions.—It is important to distinguish a falling in of the precordial region *above* the apex beat from one perceptible *at* the apex beat. The latter—that is, a systolic depression over the apex—signifies that the apical portion of the heart is adherent to the chest-wall, and it is therefore a valuable sign of previous pericarditis. But the former condition merely indicates that the heart has a more extensive contact with the chest-wall than normally; thus a falling in of the third and fourth intercostal spaces is not uncommonly observed during the systole of an enlarged and hypertrophied heart, or of a normal heart in cases of retraction of the left lung.

A systolic, heaving, expansile pulsation of the chest-wall **above the third rib** is strongly indicative of aneurysm of the arch of the aorta. Such pulsation mainly affects the upper portion of the sternum, when the transverse part of the arch is involved, but is situated to the right or left of this bone in aneurysms of the ascending or descending portions of the aortic arch. Pulsation behind to the left of the spine may also rarely be observed in cases of aortic aneurysm. It must not be forgotten that pulsation to the left of the sternum in the second and third spaces is occasionally caused by enlargement of the infundibulum of the right ventricle.

Slight aneurysmal pulsations are easily overlooked. Their detection is aided: (1.) By viewing the chest in profile as well as from the front. (2.) By palpation, especially by the bi-manual method, the palm of one hand being laid over the upper part of the sternum, while that of the other hand is laid over a corresponding area of the spine; then on pressing pretty firmly, a slight, deep-seated pulsation may sometimes be felt. (3.) By the method of tracheal tugging first described by Surgeon-Major Oliver, and since modified by Dr. Ewart. The patient is seated in a chair with his head slightly thrown back. This may be steadied against the body of the observer, who stands behind the patient, and, placing the tips of the index fingers beneath the cricoid

cartilage, exerts gentle upward pressure on the windpipe. Then if an aneurysm is so situated as to exert downward tension on the trachea, the pulsation of the aorta is transmitted to the fingers. The sensation is rather one of tugging downwards than of pulsation, and occurs with each systole of the heart.

An external **pulsating tumour** is apparent when an aneurysm has eroded and perforated the chest-wall. The pulsating prominence is seen in the neighbourhood of the second or third right interspace in cases of aneurysm of the ascending portion of the arch, but on the lateral or posterior wall of the left chest in aneurysms of the descending portions of the aorta.

Thrills.—Of endocardial thrills the **presystolic** apex thrill is the commonest, and is a valuable sign of mitral stenosis.

FIG. 115.—Dr. Ewart's Plan of Eliciting Tracheal Tugging. The observer is supposed to be standing behind the patient. (*Sansom.*)

A **Diastolic** apex thrill is also significant of mitral stenosis, but it may accompany aortic regurgitation.

Systolic thrills are occasionally met with in cases of mitral regurgitation and of aortic obstruction; in the former condition the thrill is felt at the left apex, in the latter condition in the neighbourhood of the aortic cartilage.

Friction Fremitus produced by the rubbing together of roughened pericardial surfaces may be systolic in time, but is usually both systolic and diastolic.

PERCUSSION.

The portion of cardiac surface uncovered by lung and in direct contact with the chest-wall, with the exception of a narrow strip of heart behind

the sternum, yields a dull note to gentle percussion. This is called the **area of superficial or absolute cardiac dulness.** In a healthy adult it is bounded above by the fourth left cartilage, to the right by the left margin of the sternum, and to the left by an irregular line passing downwards and outwards from the sternal end of the fourth cartilage towards the apex; its lower limit cannot be defined with accuracy, the difference between cardiac and hepatic dulness being inappreciable.

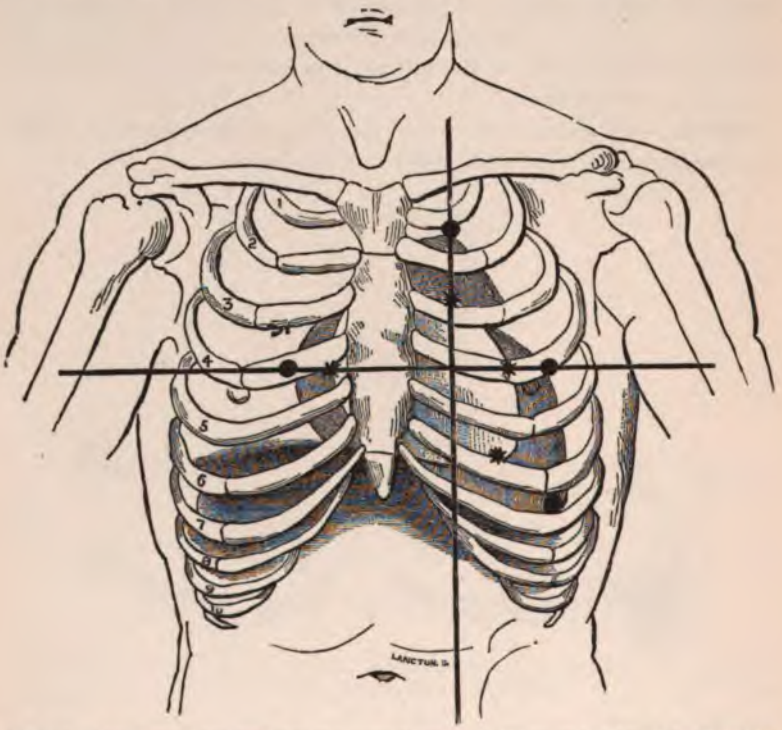


FIG. 116.—The clear space indicates the normal anatomical area of the heart, the asterisks marking the spots where percussion, in the directions indicated, first detects the influence of the underlying solid organ upon the lung resonance; in health dulness is hardly ever detectable to the right of the sternum. The dots represent the boundaries of abnormal dulness. Extension upwards indicating pericardial effusion. Extension to the right indicating enlargement of the right auricle. Extension to the left indicating enlargement of the left ventricle. (*Steell.*)

In children the area of superficial dulness is larger; its upper boundary is in the third intercostal space, and its left near the mammary line. In old age the area is smaller and somewhat lower. It is diminished by a deep inspiration. In stout persons with a short thorax and full abdomen a tympanitic note from the underlying stomach tends to obscure the cardiac dulness.

The Relative or Deep Dulness requires a forcible stroke upon the

finger, and corresponds approximately to the absolute size of the heart; it reaches upwards as high as the third cartilage, and to the left nearly as far as the nipple-line, while its right boundary extends slightly beyond that of the superficial area. In children this relative dulness begins above in the second interspace, and extends to the left a little beyond the mammary line, and usually can be more distinctly made out over the sternum than in the adult.

It is to be observed that although the heart extends to the right

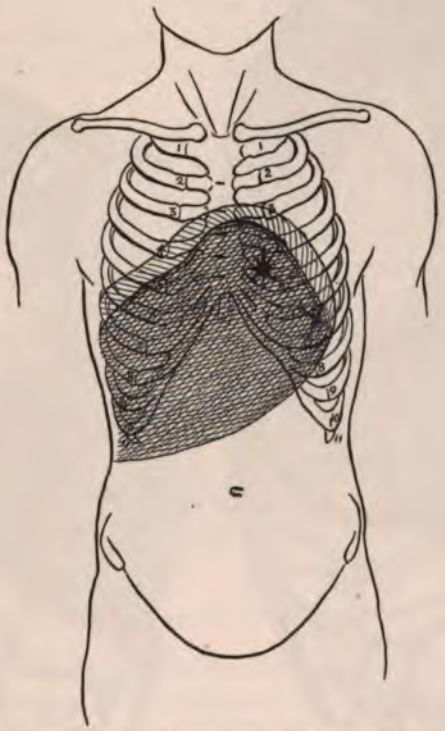


FIG. 117.—Mitral and Tricuspid Regurgitation and extreme Cardiac Enlargement in a youth aged 18. (*Dr. Leech's Case.*) The shaded area indicates the dulness yielded by the enlarged heart and congested liver—the dark shading representing the "superficial," the light shading representing the "deep" dulness.

of the sternum, *any dulness to the right of this bone*, and above that yielded by the liver, must be regarded as pathological.

The Cardiac Dulness is Increased: 1. In hypertrophy and dilatation of the heart; when the right auricle is enlarged the chief increase is to the right, when the left ventricle is enlarged, the dulness extends farther to the left and downwards, and has a more rounded outline in the region of the apex than normal.

In cases of great enlargement of the right ventricle and its infundibulum, dulness may extend upwards to the left of the sternum as high as the second cartilage.

2. In effusion into the pericardium. Here the first increase in dulness is upwards, then laterally. Very often the dull area has a

A. B.

FIG. 118.—Forms of the pericardial sac when containing (A.) 15 ounces, and (B.) 52 ounces of fluid. (*After Sibson and Byrom Bramwell.*)

pyramidal shape, the apex of the pyramid being at the second left cartilage, or even higher, while the base extends transversely from beyond the right margin of the sternum to beyond the left nipple-line. When the effusion is excessive, the area of dulness is square rather than pyramidal in shape. (See Fig. 118, B.)

In cases of pericardial effusion it is to be observed that the apex beat is not only elevated, but is situated considerably to the right of the left boundary of dulness—a distinction from enlarged heart.

3. Through undue contact of the cardiac surface with the chest-wall, either in consequence of retraction of the left lung or through forward pressure of the heart, as from a mediastinal tumour, or from some aneurysms.

The Cardiac Dulness is Diminished.—This occurs in emphysema, in pneumo-pericardium, and sometimes from tympanitic distension of the stomach.

The chief practical points to be determined in connection with cardiac dulness are the size of the right auricle, the size of the right

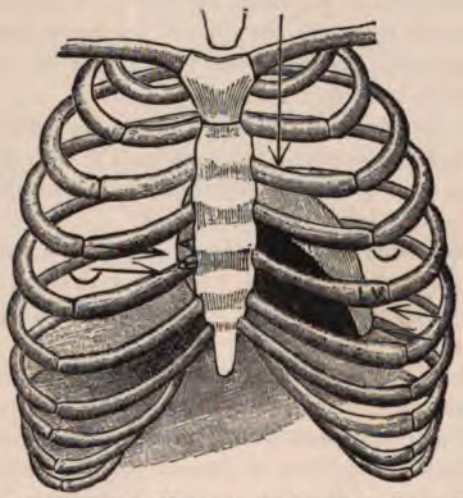


FIG. 119.—The Method of Percussion described in the Text. (*Steell.*)

ventricle and its conus arteriosus, the size of the left ventricle, and the presence or absence of pericardial effusion. The manipulations necessary to determine these points are well described by Dr. Steell as follows:—

"Percuss downwards an inch, or more if necessary, to the right of the sternum, until the liver dulness (the lung margin) is reached. Mark the spot with a copying-ink pencil. Then percuss towards the sternum not less than an inch above the level marked, and note the spot at which the modification (if any) of the lung sound, due to the auricle beneath, becomes appreciable. When absolute dulness to the right of the sternum is detected in this way, it signifies a very great degree of distension of the auricle, which has displaced the lung margin. Such distension no doubt implies imperfect systole (*systole catalectic*) on the part of the auricle.

"We next turn our attention to the left of the sternum, and endeavour to determine the size of the left ventricle. Inspection and palpation having preceded percussion, the situation of the apex-beat will probably be known. If so, let us mark the spot, noting the intercostal space in which it is situated. This spot will correspond pretty closely to the level of liver dulness already marked on the right side, or it may be a little lower. Let us next percuss from the anterior border of the axilla, or, if necessary, further outwards towards the heart, choosing the level of the apex-beat, if that has been determined by inspection or palpation; or if not, the level corresponding to rather less than an inch above the liver dulness, to the right of the sternum. Whether the situation of the apex-beat has or has not been revealed by inspection or palpation, the cardiac dulness reached at the level indicated will represent the apex of the heart closely enough for all practical purposes, and the apex is the part of the heart which extends farthest to the left. . . .

"The upper boundary of the heart-dulness is determined by percussion in a vertical direction an inch to the left of the sternum. Normally there should be no modification of lung-resonance due to the heart above the third left cartilage."

In disease, extension of the præcordial dulness in the upward direction results most commonly from pericardial effusion, but may occur in cases of extreme dilatation of the right ventricle, and especially of the conus arteriosus.

Dr. Steell has also suggested a convenient method for noting the results of percussion. "The left cartilage, to which the cardiac dulness (in the sense of lung-resonance modified by the heart) reaches, is recorded in Roman numerals, III. or II. as the case may be, while the extension of dulness (in inches) to the right and left respectively from the middle line is noted in Arabic figures placed below. Thus $\frac{\text{III.}}{3—7}$ was noted in a case of aortic regurgitation, in the last stage of the disease, and implied enormous enlargement of the left ventricle and great distension of the right auricle, with absence of pericardial effusion."

Dulness above the Third Cartilage.—This, if it be not caused by pericardial effusion, enlargement of the infundibulum of the right ventricle, consolidation of the lung, or upward displacement of the heart, is most commonly due to an aneurysm of the aortic arch, or to a mediastinal tumour. When the ascending portion of the arch is dilated, or the seat of an aneurysmal tumour, the cardiac dulness is extended upwards and to the right of the sternum. In aneurysms of the transverse portion of the arch, dulness is obtained over the upper portion of the sternum and in the adjacent intercostal spaces.

The dulness produced by a tumour is seldom so localised and defined as that produced by an aneurysm. There may be pulsation if the tumour is in contact with the aorta, but this is much less marked in relation to the area of dulness than in the case of aneurysm.

AUSCULTATION.

As a rule, it is desirable to auscultate the heart when the patient's body is upright. Sometimes, in consequence either of emotion or

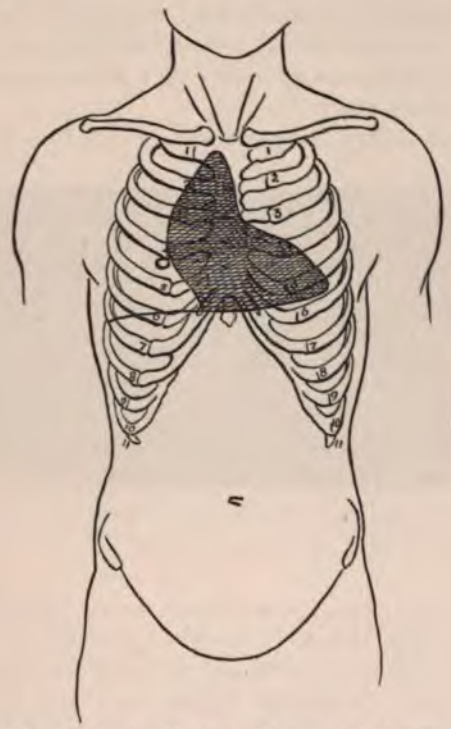


FIG. 120.—The shaded area indicates the extent of dulness in a case of aneurysm of the ascending portion of the thoracic aorta.

disease, the heart's action is too rapid for satisfactory auscultation. Then we may wait till rest in bed and the administration of digitalis, or other cardiac tonic, have steadied and regulated the heart, when the time and characters of the sounds or murmurs may be accurately determined. Sometimes, on the other hand, it is necessary to exercise the patient in order to bring out a murmur.

Normal Sounds.—On listening over the cardiac region, two sounds produced by the action of the heart are audible. One sound accompanies

the impulse, and is called the "first" or "systolic sound;" another sound follows the impulse, and is called the "second" or "diastolic sound"; it is shorter and sharper than the first sound. The second sound is most pronounced at the base, the first sound at the apex of the heart. The relative durations of the sounds and of the pauses between them are indicated in the accompanying diagram.

Position of the Valves and Auscultatory Areas.—The positions on the chest-wall at which the cardiac sounds are heard with the greatest intensity do not always lie immediately over the site of the valvular orifices. The want of correspondence is indicated in Fig. 122.

The Mitral Valve, lying much more deeply than the other valves, is on a level with the sternal end of the third left cartilage, and extends slightly behind the sternum. **The Mitral Auscultatory Area** is at the apex of the heart.

The Tricuspid Valve corresponds to a slanting line across the

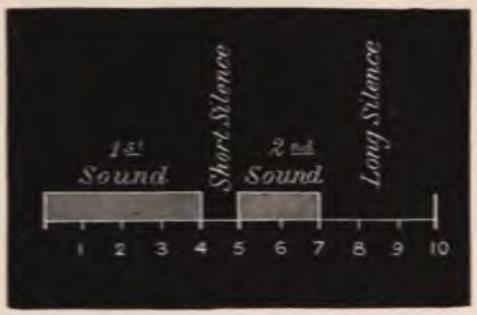


FIG. 121.—Diagram of Cardiac Cycle. The first, or systolic sound, is shown to occupy four-tenths of a second; the first, or short silence, one-tenth; the second, or diastolic sound, two-tenths; and the long, or diastolic silence, three-tenths. (*Strasburg.*)

sternum, from the third left interspace to the fifth right cartilage. **The Tricuspid Area** includes the lower end of the sternum, and the adjacent ends of the fourth, fifth and sixth left cartilages.

The two anterior **Pulmonary Valves** are placed horizontally, the left anterior cusp lies behind the upper border of the sternal end of the third left cartilage, the right one behind the sternum. **The Pulmonary Artery Area** corresponds for the most part to the position of the valves, and the third left cartilage is called the "pulmonary cartilage."

The single anterior cusp of the **Aortic Valves** lies a little lower down and nearer to the middle of the sternum than the pulmonary orifice. **The Aortic Auscultatory Area** is situated at the second right cartilage, which is therefore termed the "aortic cartilage."

Modifications of the Normal Heart Sounds—Physiological

Variations.—The loudness of the sounds varies in health with the condition of the framework of the thorax and the thickness of its coverings. Thus, in children with elastic ribs and in thin persons, the sounds are loud, and are heard over a wide area; whereas large mammæ and adiposity diminish conduction and so muffle the sounds. They are intensified during states of mental and bodily excitement. Variations in tone and duration may also be observed; thus, in young children, the sounds are shorter and clearer than in adults. Reduplication of the sounds, apart from disease, is not uncommon.

Pathological Variations.—1. **Intensification.**—The sounds are louder when the heart's action is increased from any cause, as in car-

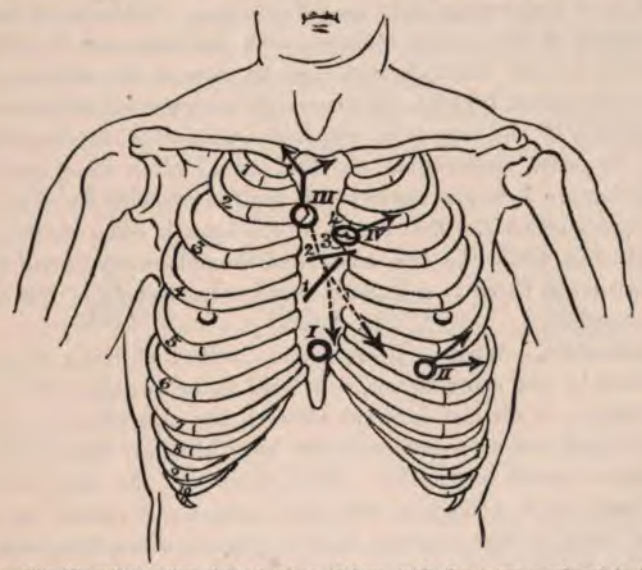


FIG. 122.—The Normal Position of the Cardiac Orifices, with the Areas employed in isolating Murmurs generated at these Orifices. 1. The tricuspid orifice; I. The tricuspid area; 2. The mitral orifice; II. The mitral area; 3. The aortic orifice (the single anterior cusp is represented); III. The aortic area; 4. The pulmonary orifice (the two anterior cusps are represented); IV. The pulmonary area (over the valves themselves). The arrows show the direction in which murmurs are transmitted. The two arrows pointing downwards and connected by dotted lines with III., the aortic area, indicate the conduction of aortic regurgitation murmurs down the left margin of the sternum and towards the left apex.

diac neuroses, and particularly in exophthalmic goitre; sometimes in anæmia, when they often possess a slapping valvular character. Increase in area of audibleness accompanies intensification of the sounds. A common cause is solidification of lung tissue.

The first sound is short and clear in dilatation, dull and toneless in hypertrophy of the heart, and often much accentuated in mitral stenosis. Accentuation of the second sound at the pulmonary cartilage is a marked feature of mitral disease, and is produced by the other causes which

increase the tension of the pulmonary circulation; it signifies hypertrophy of the right ventricle, and when the strength of the ventricle fails, the accentuation of the second sound diminishes.

Accentuation of the second sound at the aortic cartilage suggests hypertrophy of the left ventricle, and is found in association with increase of arterial pressure, as in cases of granular kidney. The accentuation is pronounced in aneurysm, also in dilatation, and in atheroma of the thoracic aorta. In these cases the second sound has often a booming or ringing character.

Enfeeblement of the Sounds occurs in cases of general debility, in degeneration of the cardiac muscle, and when the heart is unduly covered, as in emphysema or hydro-pericardium. In the latter disease the weakness of the sounds contrasts with the wide area of dulness; the sounds, too, are most distinct near the top of the sternum. In fevers, as typhus or typhoid, the degree of weakness of the first sound is a measure of the degree of impaired nutrition of the ventricular walls. In acute rheumatism the first sound often loses tone and becomes impure before a distinct soft systolic murmur develops. In aortic regurgitation the first sound at the apex is often toneless; in mitral stenosis, while the first sound and the pulmonary second sound are accentuated, there is commonly some enfeeblement of the aortic second sound.

Reduplication.—When a heart sound, instead of being single, is represented by two short sounds, it is said to be reduplicated; sometimes there is a distinct interval between the two elements of the doubled sound, but more commonly the interval is not appreciable, the reduplication being incomplete. Reduplication of the first sound is usually best heard a little to the inner side of the apex. Its most common cause is high pressure in the systemic circulation, and it is therefore a frequent phenomenon in renal disease. Reduplication of the second sound is usually most distinct in the third left space near the sternum; it is common in mitral obstruction, but occurs under a variety of other conditions.

A double aortic occasionally presents a close resemblance to friction sound.

In what is called **galloping rhythm** there are three sounds, but the accent is variable. It is best heard over the ventricles, and usually indicates failing heart muscle, as, for instance, in the advanced stage of Bright's disease.

A **metallic echo** attends the heart sounds in cases of pneumo-pericardium, sometimes in pneumo-thorax, rarely in pulmonary excavation, and occasionally in consequence of the proximity of the stomach distended with air.

Cardiac Murmurs.—A murmur is an abnormal sound heard over the cardiac region in connection with the movements of the heart; when it is dependent on changes within the heart, it is called *endocardial;* when on changes outside that organ, it is sometimes called *exocardial* or pericardial.

Endocardial Murmurs are more or less blowing sounds which replace or accompany the normal heart sounds. They are divided into **organic** and **functional**; the former class depend on some structural change at one of the orifices of the heart, but in the latter class structural change is supposed to be absent. Functional murmurs have been classed as **hæmic** when they were thought to be due to an alteration in the quantity or quality of the circulating blood; as **dynamic** when some transient irregularity in the contraction of a part of the ventricular muscle was supposed to be present.

But as the pathology of these so-called functional murmurs is not yet fully understood, and as our only object in this manual is to deal

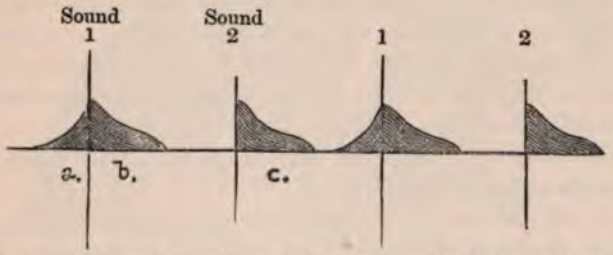


FIG. 123.—Represents the Different Rhythms of Murmurs as described in the text. *a.*, presystolic; *b.*, systolic; *c.*, diastolic. (*Steell.*)

with clinical facts, it is unnecessary to consider them separately—that is, apart from murmurs which are more certainly of organic origin.

The main facts to be determined with regard to any endocardial murmur are: (1) its rhythm; (2) its point of maximum intensity; (3) its direction of transmission.

Rhythm.—A murmur is most accurately timed by keeping the finger on the carotid or subclavian artery while listening to the heart. A murmur is **systolic** if it occupies the whole or any portion of the period from the commencement of the first sound to the beginning of the second, or if it is synchronous with the pulsation of the carotid or subclavian artery. It is **diastolic** if it occurs at any time after this period, or follows the pulsation felt in the neck. A diastolic murmur which immediately precedes the first sound or the arterial pulsation is termed *presystolic.*

Systolic Murmurs in the Mitral Area.—A systolic murmur audible in the neighbourhood of the apex beat signifies that, with each con-

traction of the heart, blood regurgitates from the left ventricle into the left auricle. The murmur is usually soft and blowing; sometimes it is harsh, and less commonly musical in quality. It is of variable length; it may replace the first sound, or follow it. In the diagnosis of the condition on which the murmur depends, **extent of conduction** is of first importance.

I. **If limited in area**, and at the most not audible behind the mid-axillary line, the first sound being heard at and beyond this limit, there is probably no great degree of regurgitation. This condition may be due:—(1.) To muscle failure, the mitral valves becoming incompetent owing to not receiving that assistance from muscle contraction which they should normally receive. The weakness of the ventricular wall may be either primary, as from chronic alcoholism or anæmia; or secondary, as for example in the later period of aortic regurgitation. Or (2.) to stenosis of the mitral orifice, when it is often the only murmur present. Or (3.) to slight structural change, as in recent endocarditis. Usually there are other auscultatory signs to aid us in forming a diagnosis, such as the association of a presystolic murmur or of an accentuated first sound in mitral stenosis; but it is impossible to diagnose between the three conditions without an appeal to collateral circumstances.

II. The murmur is conducted into the axilla, and **is heard behind at the angle of the scapula.**

(1.) In the vast majority of such cases the regurgitation depends on structural disease of the valves; for example, in rheumatic fever at an early period, a soft systolic apex murmur may be heard of limited conduction, which later, owing to severe crippling of the mitral curtains, becomes transmitted to the back.

(2.) Occasionally the reflux resulting from primary muscle failure is extreme, and then the systolic murmur is heard round to the angle of the scapula. It should here be noted that there is no necessary correspondence between the degree of valvular incompetence and that of dilatation of the left ventricle; the latter may be great when the former is slight, or *vice versa*.

(3.) Less commonly in mitral stenosis. In many cases an error in diagnosis is avoided by the presence of a presystolic murmur along with the widely conducted systolic one.

In the Aortic Area a systolic murmur of maximum intensity at the second right cartilage and conducted upwards is very common, and usually indicates obstruction at the aortic orifice, either relative or absolute.

1. In **relative** obstruction there is enlargement of the aorta, while the aortic aperture remains of natural size, or is even greater than

normal. In cases of dilated aorta, for example, fluid veins are generated by the passage of the blood through a non-constricted orifice into the wide channel beyond.

2. In **absolute** obstruction—that is, where the orifice itself is narrowed in consequence of local disease. Then the murmur tends to be harsh and prolonged; it is audible over a wide area in front and in the left upper interscapular region, and is often accompanied by a basic thrill; a diastolic murmur is often absent, and the second sound inaudible.

Such severe aortic obstructive disease is comparatively rare, and in

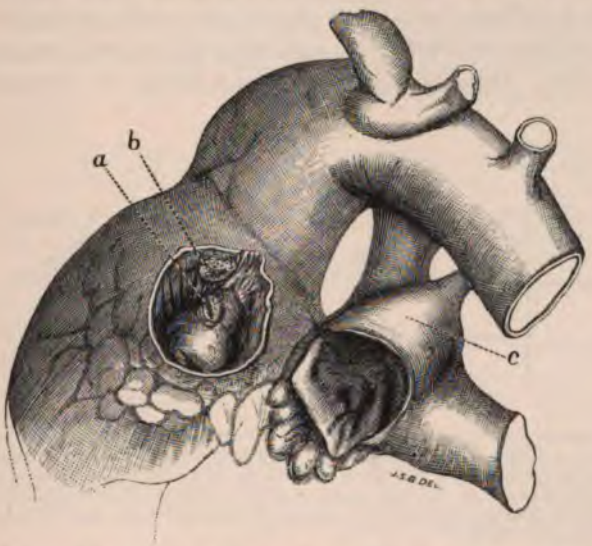


FIG. 124.—Congenital Contraction of Orifice of Pulmonary Artery from Fusion of the Valves; Foramen Ovale open. From a case of extreme cyanosis in a female who lived to the age of 22. The pulmonary valves were blended together, and formed a dome-shaped projection; at the top of this dome was a slit-like aperture (*a*), which was fringed with minute vegetations, and on the wall of the artery immediately above this aperture was a small cluster (*b*) of recent vegetations. (*Lancet*, 1884.)

most cases an aortic systolic murmur is the result either of relative obstruction or of some roughening or projection near the valvular orifice. Sometimes an aortic murmur is conducted as far as the apex, gradually diminishing in intensity. When, however, there is an increase in intensity or a change in quality near the apex, the presumption is that mitral incompetence is also present.

In the Pulmonary Area a systolic murmur occurs: (1) in anæmia; (2) in congenital pulmonary stenosis, when the murmur is loud and harsh; and (3) in some cases of cardiac dilatation. It is not uncommon apart from any obvious signs of disease.

In the Tricuspid Area a systolic murmur is often heard in cases of regurgitation through the tricuspid orifice, which occurs either as a result of dilatation of the aperture from muscle failure, or rarely from endocarditis. In many cases, however, dilatation of the right heart is unaccompanied by murmur.

Diastolic Murmurs in the Mitral Area are significant (1) of mitral stenosis, or (2) of aortic regurgitation.

1. The commonest murmur in mitral stenosis is one occurring just before the first sound, and hence called **pre-systolic.** It is peculiarly harsh and rough, and increases in intensity till suddenly ended by the first sound which is accentuated. It is limited to the neighbourhood of the apex beat, being but rarely transmitted to the left axilla. It is the only murmur which testifies with certainty to actual disease of the mitral valve. Three fallacies are to be noticed. (1.) A pseudo-pre-

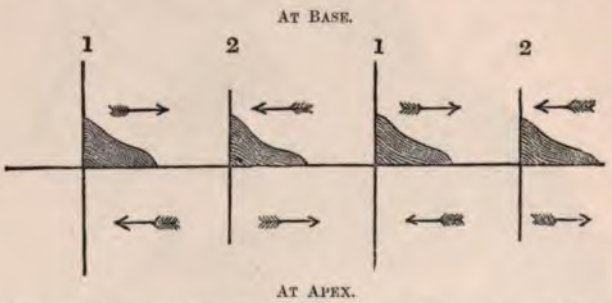


FIG. 125.—The arrows represent the direction of the current generating systolic and diastolic murmurs, according as these are formed at the base or apex of the heart. Thus a systolic murmur at the base is of obstruction mechanism, at the apex of regurgitation mechanism. A diastolic murmur at the base, again, is of regurgitation, at the apex of obstruction mechanism. The long vertical lines represent the first sound, the short the second sound, as in Fig. 123. (*Steell.*)

systolic murmur occurs very rarely in aortic regurgitation when the left ventricle is enlarged. (2.) A reduplicated first sound in Bright's disease may occasionally present a close resemblance to a pre-systolic murmur and first sound. (3.) Sometimes in mitral stenosis the second sound is absent, when the accentuated first sound following a long pre-systolic may be mistaken by a careless observer for the second sound following a systolic murmur.

A **diastolic murmur** is very often found in association with the pre-systolic in mitral stenosis; it is less harsh, however, and has a diminuendo character—that is, the intensity of the murmur lessens towards its end, whereas the pre-systolic is of crescendo character.

The diastolic murmur, heard in the mitral area, may occur alone; or it may alternate with the pre-systolic; or both being present one predominates; or both may be absent for a time. The auscultatory

phenomena of mitral stenosis are indeed remarkable for their changeable character.

2. The diastolic murmur of aortic incompetence is often conducted to the apex, rarely it is limited to that spot; in the latter case a difficulty in diagnosis may arise, but the associated symptoms of aortic regurgitation are usually present.

In the Aortic Area, a diastolic murmur heard at the sternal end of the second right cartilage, and usually still better below this, especially along the left edge of the sternum, is indicative of regurgitation through the aortic aperture into the left ventricle; if feeble, it is liable to be missed, especially when, as so often happens, it is limited to an area near the lower end of the sternum. Its quality is usually soft and blowing and its pitch high, though occasionally it is harsh or musical and very loud. It may be audible and have its maximum intensity at the apex beat.

The lesion in a large number of cases is dilatation of the aortic orifice as a part of general dilatation of the vessel; in other cases there is or has been rheumatic or septic endocarditis, or chronic thickening and shrinking of the semi-lunar cusps, as a result of frequent physical strain; or rupture of a valve; or deformity of the valves in consequence of atheroma.

The second sound is very often present, and then the murmur runs off from it. When there is accentuation of the second sound, the incompetence often depends on dilatation of the aorta. Sometimes an accentuated second sound exists for a time without a murmur, and by itself should always suggest repeated careful examinations in anticipation of a diastolic murmur. This murmur is commonly associated with an aortic systolic one, which, however, indicates relative rather than real constriction of the aortic orifice. The two murmurs sometimes produce a to-and-fro sound like that of the sawing of wood—hence called *bruit de scie*.

In the Pulmonary Area.—In mitral stenosis, and sometimes in other conditions leading to obstruction of the pulmonary circulation, there is occasionally heard at the sternal end of the third left cartilage, and for a short distance below it, a soft blowing diastolic murmur which runs off from an accentuated second sound, and when the second sound is reduplicated it runs off from the latter portion. This Dr. Steell calls the murmur of high pressure in the pulmonary artery, and considers that it is due to real regurgitation through the pulmonary aperture. Being situated near the left border of the sternum, the condition can only be distinguished from aortic incompetence by collateral evidence such as the absence of an aortic pulse, and of enlargement of the left ventricle.

Pericardial Friction.—The smooth surfaces of the healthy peri-

cardium glide upon each other without the production of any sound; but when they are roughened by disease, certain rubbing, creaking, or grating noises may be heard which are called pericardial friction sounds. These adventitious sounds occur in pericarditis, being due at the commencement to increased vascularisation or to dryness of the membrane, at a later period to exuded lymph.

Pericardial friction is usually limited in extent, but it may be heard over the whole of the præcordial region, and even to some extent beyond it. As a rule it is heard first at the base of the heart, and is also usually well heard to the left of the lower part of the sternum. It accompanies the movements of the heart, and hence has a to-and-fro character, but it is usually loudest during systole. Unlike pleuritic friction, which disappears during the stage of effusion, pericardial friction is often well heard when there is a great deal of fluid in the pericardial sac.

Occasionally friction is absent during the whole course of pericarditis. This is especially apt to occur when the exudation is purulent, probably because the vigour of the heart-muscle is depressed.

The intensity of friction depends upon the vigour of the heart's action, upon the condition of the inflammatory exudation, and to some extent upon the position of the patient.

The following additional characters serve to distinguish pericardial friction sounds from endocardial murmurs:—

1. They have a distinctly superficial character.

2. Pressure with the stethoscope may modify their quality, and render them more intense.[1]

3. They usually maintain the same tone and pitch throughout, and do not begin with an accent or shock, as is the case with endocardial murmurs; nor are they transmitted in definite directions.

4. They are associated with the movements rather than with the sounds of the heart.

5. They may shift their position or undergo changes in strength or character within a few hours.

Pericardial friction usually disappears gradually. Its cessation may be due to enfeeblement of the heart. It is important to remember that friction may be produced by very slight changes in the pericardium, which are unaccompanied by effusion or by symptoms of any kind.

Auscultation of the Arteries—In Health.—If the stethoscope be placed very lightly over the carotid or subclavian artery, two sounds resembling the heart sounds are audible. On listening over the femoral

[1] According to Ringer and Phear the character of an *endocardial* murmur is much altered by pressure with the stethoscope—the murmur becomes feebler and its pitch is raised. (*Lancet*, Feb. 10 1894.)

artery a single sound almost toneless, a sort of dull thud, systolic in rhythm, is alone audible. If pressure be made with the stethoscope, the first sound is usually at once transformed into a soft murmur, but the second sound heard in the carotids and subclavians remains unchanged. Traube has described a double sound in cases of great aortic regurgitation.

In Disease.—A systolic murmur is heard over the carotid and subclavian arteries, in anæmia, in Graves's disease, and in aortic regurgitation; and in the last condition usually whether a systolic murmur is audible over the aortic cartilage or not. On listening over the femoral artery in cases of aortic regurgitation, the normal dull thud is replaced by a more distinct sound, which may closely resemble a cardiac sound.

When pressure with the stethoscope is made over the large arteries in aortic incompetence, a systolic and a less loud diastolic murmur are usually developed.

Auscultation of Thoracic Aneurysms.—On listening over aneurysms of the aortic arch, it is more common to find altered sounds than murmurs. The first sound tends to lose tone, and may be represented by a dull thud or a mere jog or push. The second sound is often accentuated, and this sign, when the diagnosis of aneurysm, based on other signs and symptoms, is doubtful, should be regarded as one of considerable significance. This is especially the case if the accentuation is heard where normally the cardiac sounds are not distinct. Accentuation of the second sound over a circumscribed dull area in the upper part of the chest is strongly suggestive of aneurysm. The presence of murmurs may be explained by the co-existence of valvular disease of the heart, sometimes, however, they are undoubtedly produced at the mouth of a saccular aneurysm, and may be systolic or diastolic; the former being the commoner and usually the harsher, the latter the more prolonged of the two.

A systolic murmur heard to the left of the spine, apart from evidence of valvular disease, may be an important sign in the diagnosis of aneurysm of the descending portion of the aorta.

Auscultation of the Veins.—On listening over the internal jugular veins at the root of the neck in anæmic subjects, a humming sound—*bruit de diable*—is frequently heard. The murmur is *continuous*, and so differs from a murmur heard in connection with an artery. It is usually better heard on the right than the left side of the neck; it is louder when the patient is standing than when he is lying down, and becomes intensified whenever the blood-current through the jugulars is accelerated. The significance of this murmur is not great, for it is occasionally present, although feebly marked, in healthy persons, while it is absent in many cases of anæmia.

THE PULSE.

By the term pulse we indicate that alteration in the calibre of a blood-vessel which results from variations of pressure exerted by its contained blood, the pressure being regulated by the action of the heart, the elasticity of the larger blood-vessels, and the resistance in the arterioles and capillaries. A pulse may be obtained in almost any superficial artery, but as a matter of custom and convenience, we make use of the radial artery at the wrist, and, when simply *the pulse* is spoken of, it is understood to mean the radial pulse.

Methods of Examination.—The pulse may be investigated by (1) digital and (2) instrumental examination.

Digital Examination.—This method is still the most accurate and generally useful, but skill in the detection and interpretation of variations in the pulse can only be acquired by long practice and careful observation.

The first two or three fingers are to be lightly placed over the radial artery. On moving them laterally or from side to side *across* the artery, and then upwards and downwards *along* it, its direction, calibre, and the condition of its walls may be determined.

Pressure of varying degree on the vessel may now be made, usually with one finger, and first that nearest to the heart, which should be the forefinger. By this means variations due to alteration in the force or character of the ventricular systole will be evidenced by the frequency, the rhythm and the force of the pulse. The condition of the vessel between the beats should then be noted as to whether it is relatively empty or full. The degree of compressibility should also be determined by ascertaining the force required to obliterate the pulse. Further still, the general character of each pulsation must be examined as to its rise, duration and fall.

And, lastly, the pulse on one side of the body must be compared with that on the opposite side.

Thus, in a systematic examination of the pulse, the following points require investigation:—

1. The state of the coats of the artery.
2. The character of the pulse when considered as a sequence of events or *series* of beats.
3. The characters of the pulse when considered as a single event or individual beat.
4. The symmetry of the two pulses.

Instrumental Examination.—The sphygmograph has been introduced as a ready and convenient means of graphically recording the

general characters of the pulse. Several forms of the instrument have been devised. Those most frequently used are either a modification of Marey's or else the smaller ones of Dudgeon, Pond, or Richardson.

However they may vary in general form, each essentially consists of (1) *receiving part*, usually a spring or button resting on the vessel; (2) *lever*, whereby the movements of the receiving button are transmitted and amplified; (3) *recording apparatus*, a smoked paper moved by clockwork, on which the distal end of the lever records the curves which constitute the graphic representation of the pulse.

Most sphygmographs have also some means of varying the degree of pressure exerted by the receiving button on the artery. In the case of the compact and convenient instrument introduced by Dudgeon, this is brought about by means of an "eccentric," the dial of which is

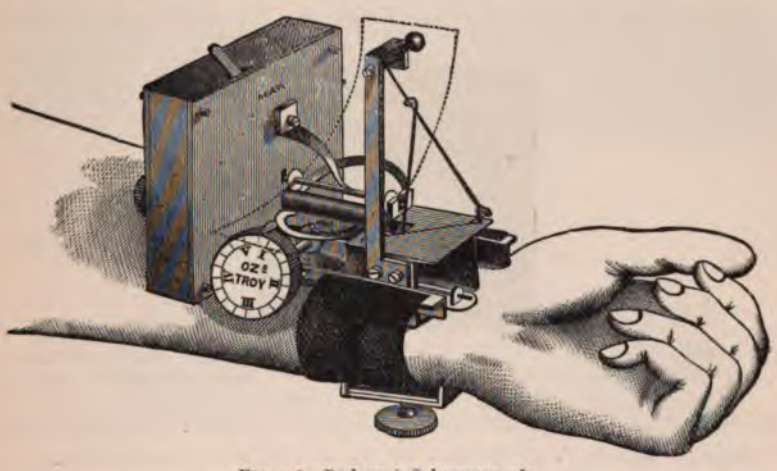


FIG. 126.—Dudgeon's Sphygmograph.

marked in ounces. While some such means of altering the pressure is most convenient, it must be clearly understood that the sphygmograph will not afford accurate measurement of the amount of pressure used. On the same pulse most different forms of tracing may be obtained by varying the amount of pressure. Hence several tracings should always be taken under varying degrees of pressure; then by a study of the series the true characters of the pulse may be determined, and the tracing or tracings in which these characters are best brought out should be kept for comparison with the results of subsequent examinations.

The chief "events" in a normal pulse tracing, together with the terms usually given to them, are indicated in Fig. 127.

The percussion wave is due partly to the contraction of the muscles

of the ventricular wall and partly to that of the papillary muscles. Roy and Adami believe that the elevation is mainly due to the action of the musculi papillares; hence they suggest the term "*papillary wave*" instead of "*percussion wave*," and they call the "*tidal wave*" the "*outflow remainder wave.*" The dicrotic or recoil wave is believed to be caused by the reflection of an impulse from the closed aortic valves.

Condition of Arterial Walls.—In patients the subjects of extensive arterial degeneration the radial artery often participates. Sometimes there is a general thickening of the vessel, in other cases irregular patches of induration can be felt, while occasionally the normal elastic artery is converted into a calcified tube.

Characters of the Pulse as a Series of Beats—Frequency.—This varies in health according to age, sex, posture, time of day and other circumstances. It is also influenced by mental emotion, by exercise, and by the administration of certain drugs. In disease the pulse is

FIG. 127.—The Normal Pulse-trace magnified. *a*, *b*, percussion up-stroke; *a*, *b*, *c*, percussion wave; *c*, *d*, *e*, tidal wave; *e*, *f*, *a*, dicrotic wave; *d*, *e*, *f*, aortic notch; *f*, *a*, diastolic period. (*Sansom.*)

accelerated in a large number of morbid conditions. In fevers the increase is usually in proportion to the elevation of temperature. A rise of 1° F. corresponds approximately with an increase of about ten pulse beats. The degree of acceleration, however, varies in different fevers, thus, with the same temperature, the pulse in scarlet fever is more frequent than in typhus. Also, a normal pulse rate may coexist with a high temperature as in typhoid. The proportional increase is greater in the child than in the adult; and there are also individual peculiarities, in some persons a slight elevation of temperature being accompanied by very considerable acceleration of the pulse.

Increased frequency also occurs in acute forms of heart disease and in chronic valvular disease when compensation fails—this is especially noticeable in mitral cases. Persistent acceleration of the pulse occurs in Graves's disease, and for a long time may be the only manifest sign of this affection.

Occasionally persistent acceleration of the pulse or paroxysmal

attacks of increased cardiac pulsation arise in the absence of any ascertainable cause. Sometimes they are related to mental excitement or to overstrain of the heart—as in the "irritable heart of soldiers." Sometimes they occur in gouty individuals, and sometimes they follow a single

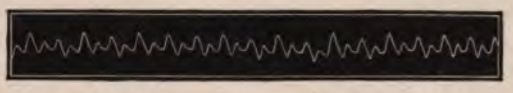


FIG. 128.—"Tachycardia;" temporary occurrence in a case of Diabetes Mellitus; pulse 200 per minute. (*Steell.*)

act of excessive exertion. In these attacks of so-called *tachycardia*, the pulse rates may be as high as 200 or even 300 per minute.

A frequent pulse is often an early sign and a striking feature in cases of osteo-arthritis.

FIG. 129.—Typical Tracing from a case of Mitral Stenosis. (*Steell.*)

Diminution in Frequency—*Bradycardia.*—A pulse of 40 is rare, but may coexist with good health. An infrequent pulse occurs after the fall of the temperature in fevers. It is also met with in cases of

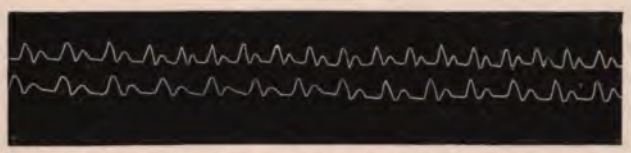


FIG. 130.—Tracing from same case as Fig. 129—the condition of low tension lasted several days. (*Steell.*)

jaundice, in fatty degeneration of the heart, and in connection with epileptic seizures; sometimes there are two heart beats to one pulse.

The frequency of the pulse is often diminished in the early stage of

FIG. 131.—Pulse in Mitral Stenosis. The tracing shows a well-marked tidal wave and the occasional occurrence of abortive beats. (*Steell.*)

meningitis, in cases of aortic stenosis, in myxœdema, in acute nephritis and in some cases of anæmia.

Equality.—In health all the beats are not only regular in rhythm,

but equal in size and of uniform force. In certain morbid conditions this regularity of volume is broken, more particularly in cases of cardiac dilatation, and in some forms of mitral stenosis. When a large wave thus alternates with a smaller one, we have what is sometimes spoken of as the *pulsus alternans*. This condition of inequality is usually associated with some variety of arrhythmia, to be next con-

FIG. 132.

FIG 133.

Show tracings taken before and after recovery from symptoms of disturbed circulation—dyspnœa, engorged liver and dropsy—in a case of mitral stenosis. (*Steell.*)

sidered. Inequality is, however, in many instances, a more reliable indication of cardiac failure even than arrhythmia.

Rhythm.—In health the pulsations of the radial artery usually succeed one another with regularity, both as regards time and force, and any persistent irregularity must be regarded in the majority of cases as an indication of disease either functional or organic.

By an **irregular** pulse is meant one in which the beats occur in a disorderly series. The pulsations occur at irregular intervals, and are

FIG. 134.—"Delirium Cordis;" temporary condition of pulse in a case of muscle-failure of the heart in a patient with Bright's disease and gout. (*Steell.*)

also usually unequal in force. Sometimes there is a certain method in the irregularity, but more frequently there can be detected no serial regularity.

Sometimes these irregularities are merely temporary, arising from slight reflex irritation, originating in many instances in the gastro-intestinal tract. Tobacco is a common cause of arrhythmia, and excessive tea-drinking is said to bring about a like condition. Persistent

irregularity of the pulse is often associated with conditions of cardiac failure, whether of muscular or valvular origin. An irregular pulse is a rarer condition in the child than in the adult; it is common in advanced life.

An **intermittent** pulse is one in which a beat is dropped or omitted

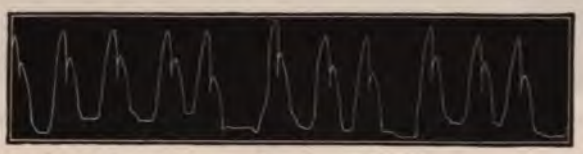


Fig. 135.—Tracing from a case of Aortic Incompetence. The patient was suffering from severe gastro-intestinal catarrh when the tracing was taken. It shows a rare form of intermission, which was perceptible to the finger. (*Steell.*)

from time to time. This may occur at regular intervals, say every eighth beat, or irregularly so that there is only an occasional omission of a beat.

Temporary conditions, such as mental excitement, digestive derangement, or excessive indulgence in tobacco may give rise to this condition.

Fig. 136.—Showing "missed beat," with succeeding unusually full beat, from a case of muscle failure of the heart, with great dilatation of the left ventricle. The systole had, no doubt been habitually incomplete for a long time preceding patient's death. (*Steell.*)

It is not infrequent in old people, and is common in the subjects of chronic gout.

Moreover, in exceptional cases an intermittent pulse may exist during the whole of a healthy life. Hence this sign by itself cannot be considered as of serious moment. Sometimes a ventricular systole is

FIG. 137.—Bigeminal Pulse; case of Aortic Incompetence; Digitalis. (*Steell.*)

too feeble to develop a perceptible pulse beat, but the sphygmograph indicates an abortive pulse wave. To the finger the pulse is apparently very slow, but the cardiac contractions present a normal frequency. The condition of *false intermission* is not uncommon in cases of mitral stenosis, especially when under the physiological action of digitalis.

Very frequently the cardiac contractions occur in couples, and between each couple there is an interval longer than natural. This constitutes

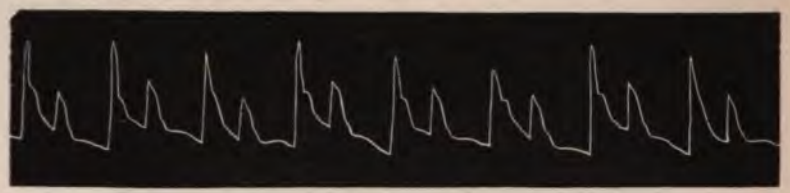


FIG. 138.—Bigeminal Pulse; also from a case of Aortic Incompetence. (*Steell.*)

the *pulsus bigeminus*, a condition which is met with in many varieties of heart disease, especially when treated by digitalis.

FIG. 139.—Bigeminal Pulse; case of "Senile Heart." (*Steell.*)

Occasionally the pulse waves occur in threes—this is the *pulsus trigeminus*. The first wave is usually the strongest, and the second stronger than the third. The *pulsus paradoxus* is another form of

FIG. 140.—Trigeminal Pulse; "Muscle-failure" of Heart; no Digitalis. (*Steell.*)

arrhythmia, in which the pulse waves become smaller or inappreciable during each inspiration. It occurs in certain cases of chronic mediastinitis, associated with adherent pericardium, but has also been

FIG. 141.—"Pulsus Paradoxus." Dr. Leech's case of Chronic Mediastinitis.

observed in other conditions, as acute pneumonia, extensive pleural effusion and aneurysm of the thoracic aorta. Dr. Brockbank has

observed the *pulsus paradoxus* in cases of acute laryngitis, and he believes the explanation "is to be found in the great variations of the intra-thoracic pressure on the heart and great vessels in the chest cavity."

Characters of Pulse as a Single Beat—Celerity.—While frequency

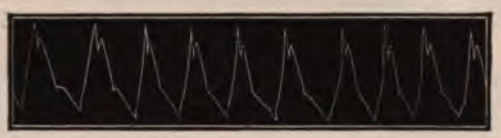


FIG. 142.—Typical Tracing from case of Aortic Incompetence of Rheumatic Origin. The deficient development of the dicrotic wave and the character of the percussion wave are the features to be noted. (*Steell.*)

refers to the number of beats taken in series, celerity or quickness relates to a single beat, and hence its character can be most readily investigated in the infrequent pulse.

In the *pulsus celer* a quick, sharp, abrupt beat is felt, usually followed immediately by an equally sudden collapse. Sometimes the

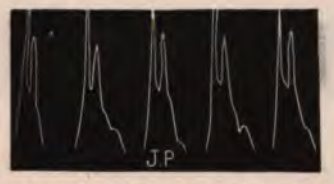


FIG. 143.—Aortic Incompetence; no Obstruction. (*Steell.*)

character of such pulse is aptly expressed by such terms as "jerky," and "slapping."

A quick pulse is especially marked in many febrile conditions and cases of cardiac dilatation. It is a special feature of Graves's disease.

A special form of abrupt pulse is met with in cases of aortic regurgi-

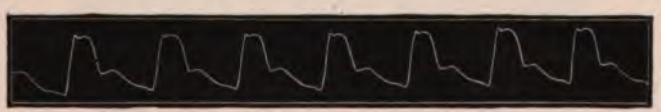


FIG. 144.—Tracing from case of Dilatation of the Aorta, with slight Secondary Incompetence of the Valves; patient æt. 56. (*Steell.*)

tation known as the "*collapsing*" or "*water-hammer*" pulse, or from the physician who first drew attention to it, as *Corrigan's*. Its characters are best brought out by raising the patient's arm above the head. The pulse beat is sudden and slapping, and is followed by an equally sudden collapse of the vessel.

The *pulsus tardus*, or slow, deliberate pulse, is the exact opposite to

the former, being characterised by the sluggishness with which the artery is distended and then relaxes. It occurs in cases of aortic

FIG. 145.—Typical Tracing of Aortic Obstruction; the curve is Anacrotic. (*Steell.*)

stenosis, in arterial sclerosis, in chronic plumbism, and as a phenomenon of old age. As a rule, the slow pulse is associated with high arterial tension.

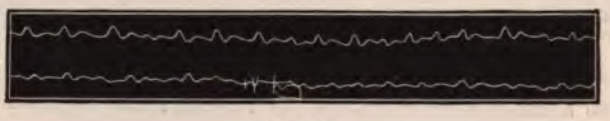


FIG. 146.

Volume and Force.—By volume is meant the magnitude or capacity of a given beat, and hence such terms as "full," "empty," "small,"

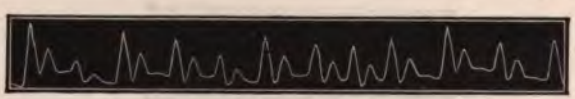


FIG. 147.

and "large" are convenient as well as correct. The force of a beat is mainly dependent upon the strength of the cardiac contraction.

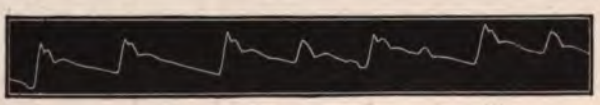


FIG. 148.

Figs. 146, 147, 148 show changes in pulse during convalescence from Alcoholic Muscle-failure of Heart. (*Steell.*)

In the *pulsus alternans* we have irregularity of volume, a large wave alternating with a small one. A large, full, soft pulse is met with

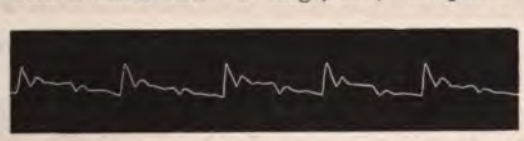


FIG. 149.—Another case of Alcoholic Muscle-failure of Heart, Bigeminal Pulse. (*Steell.*)

sometimes at the height and frequently during the decline of febrile diseases, especially if profuse sweating be present.

Tension.—By tension is indicated the degree of intra-vascular pres-

sure, and it is to be considered both during the period of distension of the artery and the period of relaxation.

This tightness of the vessel is usually estimated by the amount of digital pressure necessary to stop pulsation in the vessel. This is spoken of as the degree of compressibility of the pulse. A pulse of high tension is hard and incompressible, while that of low tension is soft and readily compressible.

FIG. 150.—Typical Pulse-tracing in a case of Acute Bright's Disease. (*Steell.*)

In many instances the sphygmograph is of the greatest service in easily demonstrating degrees of tension.

A large high-tension pulse occurs in the early stage of acute Bright's disease. A hard and very incompressible pulse, either large or small, is a striking feature of the "granular" or "contracted" kidney.

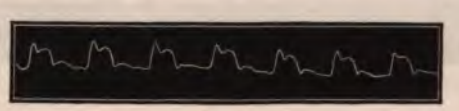


FIG. 151.—Tracing from a case of Pernicious Anæmia. Marked tidal wave; low position of dicrotic notch. (*Steell.*)

High tension is also found during pregnancy, occasionally (it is stated) in chlorosis, and to a moderate extent in many acute inflammations.

As a rule, the pulse of high tension accompanies a strongly acting heart, but it is very important to carefully observe cases where, although the pulse is quite firm, the cardiac impulse is weak and

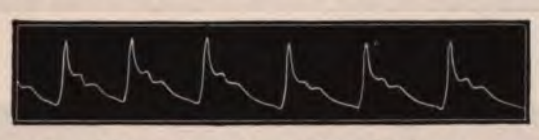


FIG. 152.—Common Type of Pulse in Simple Anæmia (Chlorosis), showing exaggerated percussion wave and small tidal wave; tension rather low. (*Steell.*)

diffused, perhaps scarcely perceptible. A hard pulse may be felt in old people in consequence of degenerative arterial changes.

A special form of low-tension pulse is the *dicrotic* pulse. For its formation three factors are usually necessary—dilatation of arterioles, sharp cardiac contraction, and unimpaired arterial elasticity. In such conditions the dicrotic wave may be so exaggerated as to be perceptible to the finger, when two beats are felt instead of one. The tracing of

such a pulse shows diminution in size or absence of the tidal wave. A pulse is said to be fully dicrotic when the aortic notch reaches the

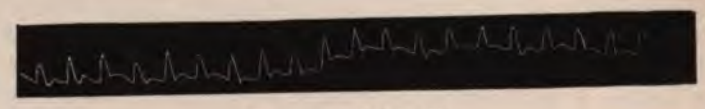


FIG. 153.—20th Oct., 1887.

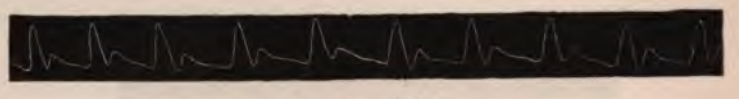


FIG. 154.—25th Oct.

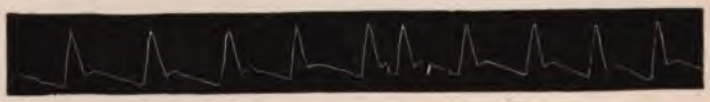


FIG. 155.—1st Nov.

FIG. 156.—4th Nov.

FIG. 157.—14th Nov.

FIG. 158.—19th Nov.

FIG. 159.—6th Dec.

Figs. 153-159 represent Pulse-tracings taken in a case of "Heart-failure" occurring during the course of Chronic Bright's Disease. The patient made a good recovery from his "heart-failure." The tracings show a progressive increase of pulse-tension. (*Steell, Med. Chronicle,* 1888.)

base line, and *hyperdicrotic* when this notch descends below the base line (see Figs. 160, 161).

An *anacrotic* pulse is one in which the tracing shows a notch in the

upstroke, the cause of which is not always the same (see Fig. 145; also Fig. 162, which shows a rare form of this variety). Mention may here

FIG. 160.—Low-tension Pulse, showing Absence of Tidal Wave. (*Steell.*)

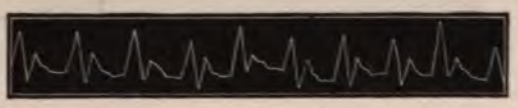


FIG. 161.—Hyperdicrotic Pulse, from a fatal case of Alcoholic Muscle-failure of the Heart. (*Steell.*)

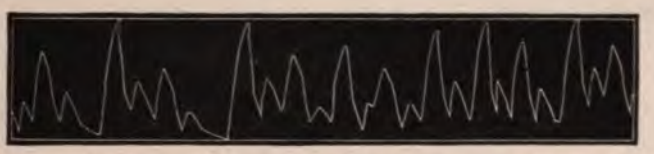


FIG. 162.—Tracing from a case of Aortic and Mitral Disease (Rheumatic), showing a form of anacrotic curve due to the commencement of the upstroke before the completion of the dicrotic wave. (*Steell.*)

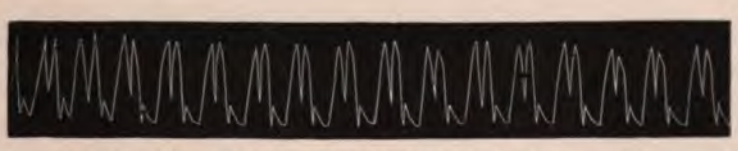


FIG. 163.—"Pulsus Bisferiens," from a case of Aortic Obstruction. (*Steell.*)

FIG. 164.—Left Pulse.

FIG. 165.—Right Pulse. Tracings of pulse in a case of Intra-Thoracic Aneurysm. (*Steell.*)

be made of the rare form of "*pulsus bisferiens*," which must not be confounded with dicrotism. It is met with occasionally in cases of aortic stenosis.

Symmetry.—A want of correspondence between the two pulses as regards time or force is commonly a result of aneurysm affecting the transverse portion of the aortic arch; it is also caused by any

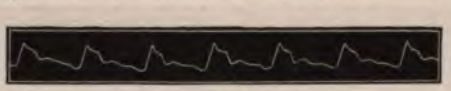


FIG. 166.—Right Pulse.

FIG. 167.—Left Pulse. These tracings show the difference between the radial pulses in a case of intra-thoracic aneurysm. Fig. 167 is characteristic of aneurysm, showing a sloping upstroke and a rounded summit. (*Steell.*)

obstruction to the arterial supply of blood to one limb, as from embolism, thrombosis, or endarteritis. It may be due to an irregular distribution of the vessels.

CHAPTER VIII.

EXAMINATION OF THE BLOOD.

As venesection is so rarely performed at the present time, an examination of the blood is usually restricted to a microscopical inspection of a few drops of blood taken from the finger.

The blood thus obtained may be mounted on a glass slide without the addition of any reagents, when a microscopical examination will reveal any marked changes in the colour of the blood, and in the shape, size and number of the red and white corpuscles. Thus the great excess of white corpuscles in leucocythæmia, or the diminution in colour and change in shape of the red corpuscles in pernicious anæmia, will be at once recognised.

But in order to make an examination with any pretence to accuracy, it is necessary to adopt some special method of preparation, or to make use of certain instruments, as the hæmacytometer, hæmoglobinometer and spectroscope.

Examination of Blood by the Dry Method.—This is a very useful method for making permanent specimens of blood. It is made as follows:—Obtain a very small drop of blood by pricking the finger; collect it on a cover slip; then with a needle very rapidly smear the small drop over the whole of the cover slip at one stroke, holding the

needle as nearly as possible parallel to the cover slip. In a few seconds this exceedingly thin layer of blood becomes dry, when the cover slip may be inverted on to a slide, and the edges cemented with paraffin; or it may be fixed to the slide with a very small drop of canada balsam, placed just at its edge.

Examination of a Drop of Blood diluted by some Fluid which fixes the Corpuscles.—For this purpose one of the following solutions should be used :—

(1.) Hayem's solution, which is composed as follows :—Sodium chloride, grm. 1 ; sodium sulphate, grms. 5 ; corrosive sublimate, grm. 0.5. and distilled water 200 grms. (2.) A 1 per cent. solution of osmic acid, or a solution consisting of one part of a 1 per cent. solution of osmic acid and two parts of a 0.75 per cent. solution of sodium chloride.

The best way to use these solutions is to place a drop of one of them on the patient's finger and then prick the finger at this spot, that is, through the drop. The blood runs into the fluid, and the mixture is readily transferred to a slide by touching it with the latter.

FIG. 168.—Poikilocytosis, from a case of albuminoid degeneration of the kidney, liver, spleen and intestine. (*v. Jaksch.*)

On placing the slide under a microscope, the condition of the corpuscles and the characters of any foreign bodies present may be accurately investigated.

Normal blood shows red corpuscles, white corpuscles, and a third class of elements—the blood plates, or Bizzozero's corpuscles. The blood plates may be well stained by the addition of a little methyl blue to Hayem's solution. They are small oval bodies, non-nucleated, and less than half the diameter of the red corpuscles; they occur singly or in groups; their physiological and pathological significance have not yet been ascertained with certainty.

The Red Corpuscles.—The shape of these corpuscles is often altered in diseases affecting the blood; this is particularly the case in pernicious anæmia. In this disease some of the corpuscles may present a normal circular outline, but others show marked deviations from the normal shape; thus they may resemble a flask, kidney, or goblet.

These irregular-shaped corpuscles are called **poikilocytes**, and the condition is spoken of as **poikilocytosis**. It is not peculiar to pernicious anæmia, but is seen in many disorders which are accompanied by marked changes in the blood, for example, in cancer, in leukæmia and in cases of albuminoid degeneration.

The size of a red corpuscle may be measured by a microscope with a micrometer eyepiece. The diameter of a normal red corpuscle varies from 6.5–9.4 μ (μ is $\frac{1}{1000}$ part of a millimetre, or 0.001 mm.). In pernicious anæmia large corpuscles from 10–15 μ in diameter, called **megaloblasts**, are often present; also corpuscles smaller than natural, called **microcytes**—some of these small corpuscles are darker in colour than normal red corpuscles, and are globular in shape.

White Corpuscles.—In health the relative number of the white compared with that of the red blood-cells varies considerably. After a full meal the proportion may be 1 : 150 or 1 : 100, while at other times it may be 1 : 350 or 1 : 600.

FIG. 169.—Leukæmic Blood, from a case of Lymphatico-splenic Leukæmia. (*v. Jaksch.*)

The blood of children contains normally a larger proportion of white blood-cells than does that of adults; also infants at the breast show a greater percentage than those fed on cow's milk.

A temporary increased proportion of white corpuscles, called **leucocytosis**, occurs in many diseases; it is common, for example, in croupous pneumonia, in many varieties of anæmia, and in cases of sarcoma. In estimating the proportion of white to red corpuscles the blood should not be examined until some hours after a meal. An enormous increase in the number of white corpuscles is met with in **leucocythæmia**, the proportion to red being as high as 1 : 10, 1 : 5, or even 1 : 2.

In this disease it is important to observe the size of the white corpuscles, for when large leucocytes are in great excess the leukæmia is of the *splenic variety*, but when both large and small leucocytes are present, and the latter preponderate, it is probable that the lymphatic glands are involved as well as the spleen—the *lymphatico-splenic type*.

Further, v. Jaksch believes that "if many corpuscles of a transitional form are found, nucleated red cells, and especially large white multinuclear corpuscles, there remains no doubt that the bone-marrow is the seat of serious changes and the disease is of the *myelogenic* type."

Occasionally colourless oblong octahedral crystals, Charcot's crystals, are found in leucocythæmic blood.

Another condition, which may be recognised by simple examination of a drop of diluted blood, is **Melanæmia**. Floating among the blood corpuscles are granules and granular masses of pigment, black, or occasionally brown or yellow in colour. More commonly the pigment particles are enclosed in cells, resembling white corpuscles. Melanæmia is often present after a severe attack of malaria; it occurs also in relapsing fever, and rarely in melanotic sarcoma and in Addison's disease.

Spirillum Obermeieri.—In relapsing fever a peculiar organism was discovered in the blood many years ago by Obermeyer. It is present during the febrile period, but disappears as the temperature falls. The organisms consist of long, spiral-shaped, delicate threads; they are six or seven times the diameter of a red corpuscle in length, and show brisk vibratile movements in the direction of their long axis. They are best seen in a drop of freshly-drawn blood without treatment with reagents, but a dried specimen may be stained with fuchsin.

The Filaria Sanguinis Hominis is found in the blood and in the urine in some cases of chyluria. The parasite is $\frac{1}{75}$ of an inch in length, and about the diameter of a red corpuscle in width. It may be seen actively wriggling about amongst the corpuscles. In searching the blood for this parasite, it must be remembered that the filariæ may be found only at night or when the patient is asleep.

Examination of the Blood for Micro-organisms.—The skin of the finger is first well washed with soap and water and a nail-brush, and then with corrosive sublimate (1 in 1000); the latter being subsequently removed by washing the finger in alcohol, and then pouring ether over it.

Two cover-glasses are sterilised by washing in corrosive sublimate, then in alcohol and ether. The finger is pricked with a sterilised needle; one cover-glass, held in steel forceps, also sterilised, is quickly brought in contact with the surface of the blood-drop. The other cover-glass is applied to the first, so as to obtain a thin layer of blood. The glasses are then separated and dried in a chamber free from dust. When dry, they are passed three times through the flame of a Bunsen's burner, and kept at a temperature of 120° C. for several hours. They are then stained with an aniline dye for several minutes, washed with sterilised distilled water, dried, and mounted in canada balsam. Methyl blue, fuchsin, or methyl violet may be used as stains.

Anthrax Bacilli, large rod-like bodies, are found in the blood in wool-sorters' disease (malignant pustule). They are stained best according to Löffler's method. The cover-glasses, prepared as described above, are placed for five to ten minutes in a solution having the following composition:—30 cc. of a concentrated alcoholic solution of methyl blue: 100 cc. of a 1 in 10,000 solution of caustic potash. The cover-slips are then washed for five to ten seconds in a $\frac{1}{2}$ per cent. solution of acetic acid. They are then treated with alcohol, dried, and mounted in canada balsam.

Tubercle Bacilli have been detected in the blood occasionally. The cover-glass preparations are stained according to Gabbet's method (see p. 166).

Malaria.—In this disease amœboid bodies are found in the blood. They were first discovered by Laveran, and his observations have now been fully confirmed. These bodies belong to the protozoa, and to a group of organisms known as hæmatozoa. They are mostly found in the interior of the red corpuscles. In some cases they contain no pigment, in other cases pigment is seen in their interior. A sickle-shaped body has been described in the red corpuscles by Laveran.

Osler gives the following varieties:—"(1.) An unpigmented hyaline body within the red blood corpuscles which displays active movements. (2.) A pigmented amœboid body within the red corpuscles, which, under certain circumstances, may alter in size and form. (3.) A segmented body, in which the protoplasm divides into a variable number of definite small spheres. (4.) Crescentic bodies, the so-called crescent, which develop within the blood corpuscles, and form characteristic and distinctive structures. (5.) Flagellate organisms, which may be seen to develop from the intra-cellular pigmented forms, or from ovoid bodies which are altered crescents. (6.) Free flagella. To the amœboid form within the red blood corpuscles, Marchiafava and Celli gave the name of 'plasmodium malariæ.'" These bodies are always present in the blood in malaria, and there is no evidence to show that they are ever present in any other disease. Golgi has shown that, corresponding to the paroxysm of ague, there is a process of segmentation.

The examination of the blood for these organisms is of great importance, especially in the irregular forms of the disease, and in cases in which the diagnosis is uncertain. For their detection an oil immersion lens is necessary. The blood may be examined unstained, or the plasmodia may be stained by a solution of methylene blue in normal (0.6 per cent.) salt solution. The point of the finger is first washed well in soap and water, then in alcohol and ether. If the staining fluid be used a drop is placed on the finger, and through the drop the finger is pricked; a drop of the mixed blood and staining fluid is then

mounted on a slide. By this method the plasmodia are stained blue.

(For description, with beautiful diagrams, the student is referred to von Jaksch's *Clinical Diagnosis*, translated by Cagney.)

Of great importance in the examination of the blood, is the estimation of the number of red corpuscles. This may be best done by Gowers' hæmacytometer. The following is Dr. Gowers' description of the instrument and its use :—

The Hæmacytometer consists of: (1.) A small pipette, which, when filled to the mark on its stem, holds exactly 995 cubic millimetres. It is furnished with an india-rubber tube and mouthpiece to facilitate

FIG. 170.—Gowers' Apparatus. A. Pipette for measuring the diluting solution; B. Capillary tube for measuring the blood; C. Cell with divisions on the floor mounted on a slide, to which springs are fixed to secure the cover-glass; D. Vessel in which the solution is made E. Spud for mixing the blood and solution; F. Guarded spear-pointed needle.

filling and emptying. (2.) A capillary tube marked to contain exactly 5 cubic millimetres, with india-rubber tube for filling, &c. (3.) A small glass jar in which the dilution is made. (4.) A glass stirrer for mixing the blood and solution in the glass jar. (5.) A brass stage plate, carrying a glass slip on which is a cell $\frac{1}{5}$ of a millimetre deep. The bottom of this is divided into $\frac{1}{10}$ millimetre squares. Upon the top of the cell rests the cover-glass, which is kept in its place by the pressure of two springs proceeding from the ends of the stage plate.

Various solutions have been employed for making the dilution. That which answers best consists of sulphate of soda, 104 grains; acetic acid, 1 drachm; distilled water, 6 ounces.

The method is as follows :—995 cubic millimetres of the sodium sulphate solution are placed in the mixing jar; 5 cubic millimetres of blood—obtained by puncturing the finger with the guarded spear-pointed needle F—are drawn into the capillary tube B, and then blown into the sodium sulphate solution. The two fluids are well mixed by rotating the stirrer between the thumb and finger, and a small drop of this dilution is placed in the centre of the cell, the covering-glass gently put upon the cell and secured by the two springs, and the plate placed upon the stage of the microscope. In a few minutes the corpuscles have sunk to the bottom of the cell, and are seen at rest on the squares. The number in ten squares is then counted, and this multiplied by 10,000 gives the number in a cubic millimetre of blood.

FIG. 171.—Gowers' Apparatus. A. Pipette bottle for distilled water; B. Capillary pipette C. Graduated tube, D. Tube with standard dilution; F. Lancet for pricking the finger.

The average number of red corpuscles in health is 5,000,000 per cubic millimetre. In a healthy adult man the number may be a little higher, in a woman a little lower, 4,500,000.

In estimating the number of white corpuscles, if these be not in great excess, it is best to count the number in fifty squares. This multiplied by 2000 will give the number of white corpuscles in one cubic millimetre. In distinguishing the white corpuscles, the following practical point is given by Gowers. If the focus of the microscope is raised so that the corpuscles become indistinct, the white ones, from their high refracting power, appear like bright points and can be easily counted.

The Hæmoglobinometer.—The amount of hæmoglobin is conveniently estimated by means of the apparatus designed by Dr. Gowers.

hæmoglobinæmia a transparent ruby red fluid takes the place of the *serum*. *On examination of normal serum a feeble absorption band is seen at the blue part of the spectrum, at the F line; it is said to be due to lutein. But with serum containing dissolved blood-pigment —hæmoglobinæmia—the absorption bands of oxy-hæmoglobin are obtained.*

Uric Acid in the Blood.—Dr. Garrod has shown that during a paroxysm of acute gout, uric acid may be detected in the blood. Two drachms of serum furnished by the blood after coagulation, or obtained from a blister, are placed in a glass dish, and slightly acidulated with acetic acid; a fine linen fibre is placed in the fluid, and the glass dish, protected from dust, is set aside in a warm place. In a few hours the fluid gelatinises by evaporation; then if the thread be examined under the microscope, it will be found to be encrusted with crystals of uric acid. But this process yields no crystals when the serum is obtained from a healthy person. An excess of uric acid in the blood has been detected by v. Jaksch also in anæmia, pneumonia, pleurisy, renal and cardiac disease.

CHAPTER IX.

EXAMINATION OF THE DIGESTIVE SYSTEM AND OF THE ABDOMINAL ORGANS.

THE TONGUE.

The chief objective characters presented by the tongue in disease may be classified under the headings of size, shape, colour, condition of surface, and movements.

Size.—A swollen, teeth-marked tongue during a course of mercury is one of the earliest signs of mercurial salivation; such a tongue may result from iodide of potassium, especially in patients who have been previously subjected to a course of mercury, and is also seen in some varieties of dyspepsia. It is broad and teeth-indented in anæmia, and sometimes in Bright's disease. In idiopathic glossitis, as well as in that resulting from mercury, small-pox, &c., the tongue may be so large as to protrude from the mouth. It presents moderate enlargement in cases of venous obstruction, as from mitral disease, aneurysm of the thoracic aorta, and rarely from severe inflammation of the throat. It may become swollen during an attack of urticaria, and sometimes in severe cases of pemphigus. Hypertrophy of the tongue is met with as a

fur is usually deposited in two lateral strips, the tip and edges of the tongue, which are redder than normal, and often the middle line, being free from deposit; white at first, the fur soon becomes of brownish tint. In intestinal catarrh a greyish gummy fur is common. The tongue often gets quickly coated with a thick, foul fur after an attack of cerebral hæmorrhage, or an epileptic fit. A particularly nasty, furred, teeth-indented tongue occurs two or three days after severe hæmorrhages, whether from stomach, lungs, or bowels. In diphtheria a thin brown fur is often present; it is also to be noticed that diphtheritic membrane may form on the tops of the papillæ at the back of the tongue.

It is important to remember that many furred tongues are largely so because no attrition has removed the fur; this is especially the case in acute febrile diseases. Deficient attrition, too, is the chief cause of **unilateral furring**, for the conditions with which the latter is associated are those which tend to restrict the movements of the affected side. Amongst such conditions may be mentioned absence of teeth on one side, the presence of an ulcer or the jagged edge of a carious tooth; also paralysis of one side, as in hemiplegia.

Dryness.—The tongue tends to get dry in the later stages of all severe acute febrile diseases; this is particularly the case in typhus, typhoid, pneumonia and pyæmia. Here must be noted the part that keeping the mouth partly open in fevers plays in causing evaporation and tending to make the tongue dry and baked. Towards the middle of the second week in typhus the tongue becomes small, baked and black; and in typhoid at the end of the second week there are lateral strips of dry yellowish-brown fur, and transverse and longitudinal cracks on the surface of the tongue. Dryness of the tongue is often associated with low muttering delirium; and in chronic diseases commonly suggests a fatal termination. But it must be borne in mind that dryness of the tongue is not uncommon in the aged, apart from illness. A red, clean, dry, cracked tongue is seen in chronic dysentery.

Desquamation.—In an acute illness one of the earliest indications of improvement is a tendency to moisture, together with a thinning and recession of the fur at the tip and edges, of a previously dry, brown, furred tongue. Sometimes the fur comes off in flakes, leaving round or oval smooth patches; this occurs in measles, typhoid, and in infantile gastric catarrh. The so-called "**mapped tongue**" is a tongue of erratic desquamation with slight epithelial proliferation by the sides of the desquamated areas.

Papillæ.—In addition to the undue prominence met with in gastric catarrh and in scarlet fever, the papillæ are especially affected (and chiefly the filiform) in ichthyosis linguæ—in this disease, called also

extreme fissuring which sometimes occurs as a consequence of the prolonged administration of iodide of potassium. Small wounds and scars—with or without a local glossitis—from biting the tongue are met with in epileptics, but not in the subjects of hysterical attacks.

Adhesions may be congenital, are often the result of ulcerative processes, and firmly bind down the tongue in cases of epithelioma. In "tongue-tie" the movements of the anterior part of the organ are restrained either by a short frenum or by one attached further forwards than usual.

Disorders of Movement.—**Spasm.**—Spasmodic deviation of the tongue on protrusion has already been mentioned as occurring in hysteria (see page 56). It may also follow irritation of the fifth nerve. The tongue, too, participates in the spasms associated with epilepsy and chorea.

Tremor of the tongue is a valuable sign of chronic alcoholism; it occurs also in cases of mercurial poisoning, in general paralysis of the insane, and occasionally in paralysis agitans. After damage to the hypoglossal nerve, fibrillary tumours may be observed on the side of the tongue which is undergoing atrophy.

Paralysis.—Unilateral weakness of the tongue, causing deviation on protrusion towards the affected side, is of common occurrence in hemiplegia; it may also occur in consequence of damage to the hypoglossal nerve either inside or outside the skull, and then paralysis is followed by atrophy of the affected muscles.

Unilateral paralysis of the tongue in association with paralysis of one vocal cord and half the palate on the same side, points to a lesion at the surface of the medulla, but may be due to a tumour in the neck.

Bilateral paralysis indicates degeneration of the hypoglossal nuclei, and usually forms a part of bulbar paralysis, the lips and throat being also affected. When the paralysis is complete, the tongue lies motionless and atrophied within the mouth, its mucous membrane being thrown into irregular folds, and articulation and mastication are more or less impaired. Minor degrees of weakness are shown by an inability to raise the tip of the tongue towards the hard palate or towards the upper lip after protrusion.

THE TEETH.

The time at which the twenty milk or temporary teeth are cut is somewhat variable; the numbers in the accompanying table represent the average months, the *lower* central incisors, as a rule, being the first to appear.

Hutchinson, is, when typical, pathognomonic of hereditary syphilis ; the following points are to be carefully observed. The deformity affects the **upper, central, permanent incisors** most frequently, and these alone are characteristic. They are atrophied ; pegged, the width of the cutting edge being narrower than that of the tooth close to the gum ; and notched. A typical tooth presents a single notch, or a lunula-shaped edge (see Fig. 173), which readily becomes a notch. Children who have such teeth are also often the subjects of symmetrical deafness and interstitial keratitis.

Pitted and Rocky Enamel.—Care must be taken to distinguish the teeth of hereditary syphilis from teeth which exhibit imperfect calcification of the enamel.

Deficiency in the enamel produces lines, furrows, or pits on the surface of the permanent set of teeth ; and it is especially to be observed that the defects are horizontal, the lines or grooves running

FIG. 173.—Teeth of Child the subject of Hereditary Syphilis, showing notching of upper central incisors.

transversely across the teeth, and the pits being arranged in horizontal series. The incisors, canines and first molars are usually most markedly affected ; they may be studded with pits like a thimble, and in extreme cases the enamel is almost entirely absent. Teeth of this description are common in children who are the subjects of lamellar cataract, and who have suffered from convulsions in infancy. Mr. Hutchinson believes that the dental defect is due to stomatitis set up by the abuse of mercury in infancy. Other authorities regard it simply as an expression of defective nutrition, the result of any depressing influence in early life.

Loosening of the Teeth often results from the use of mercury, and sometimes from taking iodide of potassium. The teeth become loosened, too, in scurvy, in gangrenous stomatitis, and in diabetes ; and sometimes after measles and scarlet fever, when necrosis of the jaw may also ensue.

Grinding of the Teeth, commonest by far in children, points, as a

rule, to some form of gastro-intestinal irritation, of which that associated with the presence of worms is one. But it also occurs, apart from gastro-intestinal irritation, in children of neurotic temperament, in whom it may lead to bevelling down of the edges of the teeth. Rarely grinding of the teeth is met with in the adult in connection with rheumatism, gout, and lithæmic conditions.

THE GUMS.

The gums often show a red line in phthisis. In lead-poisoning it is common to find a blue or black line along the margin of the gums, but this is absent when the teeth are kept clean. Sometimes only a single row of black dots is visible; when indistinct, the blue line is brought out by the administration of iodide of potassium. Similar purple or blackish stains may be seen on the inside of the lips. In measles the gums are reddened, and often swell and bleed; white films are also constantly seen, as if the surface had been painted with nitrate of silver.

The gums are pale in anæmia, reddened and ulcerated in the various forms of stomatitis. They are tender and spongy in mercurial salivation and in scurvy; the swelling is especially and primarily around teeth, and does not affect parts where there are no teeth and no stumps.

Sordes.—Thin yellowish or thick brown or black crusts occur in typhoid conditions. They are found on the exposed surface of the teeth and lips. In hæmorrhage from the mouth, black crusts are seen on the gums and teeth.

THE MUCOUS MEMBRANE OF THE MOUTH.

Colour.—It is pale in anæmia, yellowish in jaundice, livid in cyanosis, and presents local alterations in stomatitis. In scarlet fever the mucous membrane is a bright red; in measles a patchy redness is seen on the hard and soft palates and on the inside of the cheeks; in purpura there are purplish spots; and in small-pox the characteristic eruption is often perfectly distinct.

In Addison's disease, brownish or bluish-black stains and streaks may be visible on the buccal mucous membrane. In lead-poisoning, too, blue stains are occasionally present on the inside of the cheeks.

In **thrush** opaque white soft specks are seen on the tongue, and the mucous membrane of the cheeks and lips; they look like particles of milk curd, but cannot be wiped off, though they are less adherent at a later stage; usually they are surrounded by a red areola, and when scraped off the mucous membrane is left raw and red, or there may be a shallow ulcer. It is most commonly met with in hand-fed babies,

being an expression of oral catarrh, produced by sour milk or other fermentable food. In neglected atrophic infants not only specks but large white patches are seen, and in bad cases the whole of the mucous membrane is covered with a white or grey membrane. Such a condition is of grave import. In adults thrush is met with towards the end of exhausting disease, especially tuberculosis.

If a scraping be placed on a glass slide with a drop of liq. potassæ or glycerine, and examined with the microscope, the spores and threads of "oïdium albicans" will be seen, together with squamous epithelium, leucocytes and detritus. The spores are oval, sharply contoured little bodies, and are often attached by their ends to form groups of two or three. The threads are branched and jointed at somewhat long intervals; at the end of each limb two polar highly refracting granules are usually visible.

White patches due to diphtheritic membrane are in rare cases present in the interior of the mouth.

Ulceration.—In teething-infants not only redness and swelling of the gums may be present, but also small shallow ulcers, or in an earlier stage vesicles, with a red areola round the base.

In older children, often rickety and ill nourished, one sees larger irregular greyish ulcers, which affect chiefly the gums, but may spread to the tongue, cheek and lips; the gum adjacent to the ulcer is red, swollen, and bleeds easily, the teeth loosen, and the breath is foul and salivation copious.

Also in young children debilitated from any cause, but especially from measles, gangrenous stomatitis may occur; here a greyish ulcer is detected in the mucous membrane of the cheek, corresponding to a hard spot in the substance of the cheek, or the ulcer may be on the gum, especially at its junction with the cheek, the saliva is bloody, and the breath has a gangrenous odour. A brownish or black slough soon forms, and when it separates, a ragged ulcer is left, which spreads rapidly, and may affect the whole side of the mouth; boring through the cheek it meets the external ulceration, and thus there may be a hole right through the cheek. The gums being destroyed, the teeth are loosened and the jaw bone is necrosed.

Bleeding from the cavity of the mouth does not occur so often as from the nostrils. It is met with in stomatitis, scurvy, purpura and in hæmorrhagic exanthemata. In the hæmorrhagic diathesis, also sometimes in leucocythæmia and in lymphadenoma, extraction of a tooth may cause dangerous hæmorrhage.

organisms are found in great numbers; this is especially the case when decomposition occurs, as in scurvy and mercurial poisoning.

THE SOFT PALATE, FAUCES, AND PHARYNX.

In order to make a satisfactory examination of the throat, two things are essential, a good light and depression of the tongue. Some patients can voluntarily depress the tongue and so expose the back of the throat; others only by taking a deep inspiration, or by saying "Ah"; but in most cases it is necessary to introduce a spatula or the handle of a spoon (or in children the finger), in order to depress the tongue and obtain a complete view of the parts behind. When good daylight is not obtainable, the throat may be well illuminated by the simple plan suggested by Henoch; a taper or candle, with a bright tablespoon behind it, is held in one hand, while the tongue is depressed with the other hand, when it will be found that the concave side of the spoon acts as a good reflector.[1] To inspect the superior or nasal portion, and the inferior or laryngeal portion of the pharynx, a laryngeal mirror is necessary; and digital examination is also often required.

Colour.—The natural pale red of the uvula, fauces and posterior wall of the pharynx is deepened in all inflammatory conditions whether of a simple or specific nature. A general increase in redness is seen in ordinary catarrh, or in that due to measles, rötheln, or typhoid; in scarlet fever the colour may be either a bright or dark red; in erysipelas it is often purple and shining. In the second stage of syphilis a sharply bounded uniform redness is frequently present. A local increase of colour is seen in tonsillitis, mainly over the inflamed tonsil; in granular pharyngitis the openings of the follicles are reddened, and in herpetic sore throat the mucous membrane around the vesicles is affected. In retropharyngeal abscess the posterior wall of the pharynx is red and prominent. In tubercular cases the pharyngeal wall is usually pale and often exhibits a fine network of injected vessels, with an intervening mucous membrane of purulent aspect. A patchy redness frequently precedes ulceration. In post-nasal catarrh the soft palate is often injected, and contrasts markedly with the paler mucous membrane of the hard palate.

Swelling.—Tumefaction is often general and uniform, affecting the soft palate and fauces equally; in many cases, however, one or other part is picked out, thus in tonsillitis the tonsils may both project as red globular masses which may meet and become flattened by mutual pressure. Quinsy or abscess of the tonsil is at first a one-sided

[1] A match-box with one side concave and a holder for the lighted match is now sold, and gives very good illumination.

affection, all the anterior arch of the palate is bulged forward, and fluctuation may be felt on introducing the finger. In relaxed throat and in œdema laryngis, the uvula is apt to be elongated and swollen, and by projection into the pharynx may excite vomiting, or by hanging down into the larynx may tend to suffocation.

Elevations.—**Vesicles** occur in herpes of the pharynx, and may be so numerous and close together as to simulate a diphtheritic membrane; a herpetic eruption on the skin or on the genitals sometimes alternates with the pharyngeal one. The vesicles of chicken-pox may also affect the throat. Sometimes the soft palate is covered with small translucent granules; this occurs in influenza, also as a chronic affection, when it is often associated with post-nasal catarrh.

Pustules may be found in small-pox, from the third to sixth day of eruption.

Adenoid Masses about the size of millet seeds are met with in the vault of the pharynx, and larger masses at the root of the tongue; in the former position they give the mucous membrane a granular appearance (clergyman's sore throat). On introducing the finger behind the soft palate small vegetations may be recognised by their velvety feel, while the sensation of large ones has been compared to that of a bag of worms. In a characteristic case the aspect of a child who suffers from adenoids is peculiar: the expression is vacant, the mouth half open, the face elongated, and the nose narrow. There is noisy breathing, and usually some impairment of hearing.

Tumours.—Papillomata are the commonest; gummata occasionally develop under the mucous membrane of the posterior wall; cancer is very rare.

Calculi, composed of phosphate and carbonate of lime, sometimes project from the follicles of a tonsil.

Exudation varying in amount is seen in nearly all throat affections. Yellowish white spots of inspissated secretion, projecting from the follicles of the tonsils, are common in tonsillitis whether simple or scarlatinal; and in granular pharyngitis caseous worms can often be easily expressed from these follicles. The above mucoid, purulent, or caseous materials may be readily removed, and leave a more or less intact surface, but in diphtheria the whitish specks or patches of membrane, which are visible at the back of the throat, and which mainly affect the palate and uvula, are adherent, and when forcibly detached leave a raw and often a bleeding surface. Ulcers covered by secretion, and patches of thrush on the fauces may present a close resemblance to diphtheritic membrane; but a careful inspection will show that the patches of true diphtheria are elevated above the surface, whereas those formed by ulceration are usually slightly depressed;

and in the case of thrush the microscope will reveal the parasitic growth.

At the same time it must be admitted that the diagnosis of diphtheria cannot always be positively determined from throat inspection alone. When the membrane lines the edges of the uvula and the palatine arch, the diagnosis is easy, but when greyish or yellowish patches of exudation are limited to the surface of red swollen tonsils, there may be considerable difficulty in deciding whether they constitute true membrane or are merely the secretions of an inflamed and ulcerated mucous membrane, as in the case of scarlet fever. In doubtful cases of this kind the diagnosis has to be based on concomitant phenomena; thus enlargement of the superficial cervical glands out of all proportion to the apparent intensity of the faucial affection, a moderate or low temperature, and the presence at an early period of albumen in the urine would be indicative of diphtheria; while slight enlargement of the glands, absence of albuminuria, the presence of a high temperature and rapid pulse would suggest that the throat condition depended either on scarlatinal or on some other poison than that of diphtheria.

Ulceration.—Small superficial ulcers are found on the tonsils in acute tonsillitis, in scarlet fever, in diphtheria, in ulcerated sore throat, and occasionally as a result of catarrhal or follicular pharyngitis. They also follow the rupture of herpetic vesicles. Shallow ulcers form on the posterior pharyngeal wall in catarrhal pharyngitis; on the mucous patches of secondary syphilis; also in tertiary syphilis when they are often serpiginous in outline, have irregular jagged edges, and occupy mainly the soft palate. In phthisis, lenticular ulcers occur on the lateral walls and spread to the posterior wall of the pharynx, to the soft palate, and to the roof of the mouth; they have greyish caseous floors, and thick congested edges, and small grey nodules are often to be seen in the immediate neighbourhood.

Deep ulceration occurs in severe scarlet fever, in diphtheria, in small-pox, and very rarely as a result of cancer. In tertiary syphilis deep non-symmetrical perforating ulcers are common; they eat away the soft palate, uvula, fauces, and back of the pharynx, laying bare it may be the bones of the palate or vertebræ. Sometimes they lead to cicatricial contraction of the pharynx. Such destructive ulceration occurs in hereditary as well as in acquired syphilis; and it may be safely asserted that most chronic ulcers of the tonsils and soft palate are of syphilitic origin.

Gangrene of the fauces may occur in bad cases of scarlet fever, diphtheria, or small-pox, and very rarely from the mere intensity of ordinary inflammation.

THE ŒSOPHAGUS.

Anatomical Relations.—The œsophagus begins at the cricoid cartilage opposite the sixth cervical vertebra. It lies to the left of the spine down to the fifth or sixth dorsal vertebra, then it crosses to the right side, but finally turns again to the left side, and passes through the diaphragm to open into the stomach at about the level of the tenth or eleventh dorsal vertebra.

The length of the œsophagus in the adult is from nine to ten inches. In passing a sound the distance from the incisor teeth to the cardiac orifice of the stomach is about fifteen or sixteen inches. The left bronchus crosses the œsophagus opposite the union of the fourth and fifth dorsal vertebræ, or at a distance of about eight inches from the incisor teeth. Above this point the œsophagus is in relation with the trachea and the recurrent laryngeal nerves; below with the bronchial glands, the aorta, pleuræ and pericardium.

Examination of the Œsophagus.—In investigating a case of suspected œsophageal disease, symptoms are usually of more value than the results of physical examination. As to the latter, careful palpation with a sound is of primary importance; inspection and auscultation are occasionally useful, but percussion is not required. In all cases a thorough physical examination of the thoracic organs is essential, and careful observations should be made with regard to the general nutrition of the body.

Inspection.—Rarely the œsophagus may present a sac-like enlargement in the neck, usually on the left side; during meal times this diverticulum will enlarge and give a dull note to percussion, and when emptied by compression rumbling or splashing noises may be heard. A very large sac sometimes presses on the trachea and causes choking sensations.

Palpation.—Puffiness of the neck, and a sense of crepitation to the fingers, owing to the presence of air in the subcutaneous tissues, may follow a sudden rupture or gradual perforation of the thoracic portion of the œsophagus.

In rare cases the right radial pulse has been found to be much weaker than the left pulse, and especially during the act of swallowing; this is explained by the origin of the right subclavian from the left side of the arch of the aorta, and its passage to the right between the spine and the œsophagus.

Diagnosis is often much aided by the introduction of a sound or bougie. This, which should not be thicker than three-quarters of an inch, is first warmed, and then moistened with glycerine. It should

be passed to the posterior wall of the pharynx, and then pushed gently onwards, the patient's head being inclined forwards to keep the bougie out of the larynx. During its introduction the following points should be observed:—

(1.) Whether there is any **pain**, and whether it is limited to a particular spot; this would indicate inflammation, which is usually caused by local irritants, or ulceration, which is commonly the result of direct injury from some foreign body.

(2.) Whether any **hindrance** is encountered. Obstruction is usually due to spasm, which is often set up by the sound itself, especially in hysterical subjects. When the passage of the sound is resisted, it should be allowed to rest for a few seconds, and then again pushed gently on, but if still hindered a smaller sound should be tried. The **size** of a stricture may be estimated from the diameter of the sound which passes through it, its **site** by the length of sound introduced, measuring from the incisor teeth. The most common cause of organic stricture is cancer, the calibre of the tube being narrowed to some extent by thickening of, and by nodular projections from its wall. In cancer, however, there is not usually much post-mortem evidence of stricture, and the obstruction during life is chiefly due to paralysis of the wall at the seat of malignant ulceration, as well as to a spasmodic contraction of the part above. The most common site is probably near the bifurcation of the trachea. Other causes of stricture are cicatrices, following the ulcerations produced by swallowing acids or alkalies, or possibly by syphilis; the tube may be also narrowed by outside pressure as from enlarged glands, an aortic aneurysm, or a mediastinal tumour.

(3.) Any material adhering to the sound should be examined microscopically for evidence of blood, pus, cancer-cells or nests, thrush-fungus, &c.

(4.) Sometimes the lateral movements of the sound are felt to be very free; this indicates dilatation of the tube, which may occur either above or below a stricture, and also more diffusely in cases of paralysis.

Precautions.—(1.) Before passing an œsophageal bougie, the presence or absence of an aneurysm of the thoracic aorta should always be considered; and in obscure cases the possibility of the obstruction in the œsophagus being due to compression from an aneurysm should be borne in mind, for the arterial and œsophageal coats may form but a thin partition to be easily broken through by the forcible pushing of a bougie, when fatal hæmorrhage would occur. Sometimes a pulsating movement of the sound gives a warning to the physician.

(2.) There is less risk of the sound entering the larynx, but this may occur when the epiglottis is paralysed and when there is anæsthesia of the laryngeal mucous membrane; then there is great danger, for no

cough warns the operator. If in such a case dyspnœa follows the introduction of a sound, this should be at once removed; and if re-introduced, the forefinger of the left hand should first be passed far back in order to reach and hold the paralysed epiglottis.

(3.) Occasionally bony thickening of the rings of the trachea or a congenital stricture of the pharynx furnish obstacles to the passage of the sound, and tend to divert its course into the larynx.

(4.) Sometimes the obstruction is constituted by a diverticulum in the œsophagus, and there may be considerable difficulty in finding the natural channel.

Auscultation.—Normally, on listening with the stethoscope to the left side of the neck, a short gurgling sound, or a series of gurgles,[1] is heard during the act of swallowing, and a similar but fainter sound is also audible lower down on the left side of the six upper dorsal vertebræ. By keeping a finger over the hyoid bone, the time between the commencement of swallowing and the sound heard in the back may be estimated; this interval will be found to be longer, and the gurgling sound to be more prolonged and weaker than normal, in cases of stricture—the œsophagus being auscultated below the stricture. On listening in front over the space between the xiphoid cartilage and the left costal arch, two noises are heard in health—one at the commencement of swallowing and another six or seven seconds later; the latter is more delayed and more prolonged in paralysis of the œsophagus.

THE ABDOMEN.

Divisions and Contents.—For descriptive purposes the abdomen is conveniently divided into nine unequal regions by four lines, two of which are horizontal and two vertical. The upper horizontal line is drawn across the body between the lowest points of the costal arches, the lower one between the anterior superior spines of the iliac bones. The two vertical lines are drawn upwards from the middle of Poupart's ligament. The regions thus marked out are shown in the accompanying diagram. The three upper ones are bounded above by the diaphragm.

The **Right Hypochondrium** contains the right lobe of the liver, with the gall bladder and the hepatic flexure of the colon. Deeper in this region lie the first and second portions of the duodenum, and still deeper the supra-renal capsule and the top of the right kidney.

In the **Epigastric** region are situated the body and pyloric end of the stomach and the left lobe of the liver; behind the stomach are the

[1] These sound like bubbling râles, and, when heard on listening over the upper part of the interscapular region, may lead a careless observer to diagnose disease of the lungs.

hepatic vessels, the pancreas, the coeliac axis, and the semi-lunar ganglia.

In the **Left Hypochondrium** are the cardiac end of the stomach, the spleen, the splenic flexure of the colon and, more deeply, the top of the left kidney with its capsule.

The **Lumbar** regions are occupied by the kidneys, the colon and portions of the small intestine.

Along the upper part of the **Umbilical** region the transverse colon passes, and deeper than this is the third portion of the duodenum; but the greater part of this region, and of the **Hypogastrium**, is filled by the coils of the small intestine, together with the mesentery, vessels and glands. The aorta divides opposite to and a little to the left of the navel. A distended bladder or a gravid uterus also rises up into the hypogastrium.

FIG. 174.—Showing the Anatomical Regions of the Abdomen.

In the **Left Iliac** region is placed the sigmoid flexure of the colon, in the *right*, the cæcum.

Inspection of Abdomen.—The abdomen may present a perfectly healthy appearance, even when one of its organs is extensively diseased; but as a rule disease within its cavity is accompanied by some change—it may be a slight one—in the colour, form, or movements of its surface. On the other hand, the abdomen may look abnormal, when its contents are healthy, in consequence of some general disease, or of a lesion in another part of the body.

Whenever possible the patient should be examined in the standing posture, the abdominal walls being carefully inspected and palpated behind as well as in front. This method is of great value, especially as regards the question of symmetry, and should always precede examination of the patient when lying down.

Shape and Size.—In health the form of the abdomen varies greatly in different individuals. It is more protuberant in women and children than in adult males, while in old age it often becomes very small and shrunken.

A **Uniform General Enlargement** may be due :—

1. To a fatty condition of the abdominal walls or of the mesentery; or to œdema of the abdominal walls.

2. To ascites, *i.e.*, an effusion of liquid into the peritoneal sac. In ascites the shape of the abdomen varies with change of posture, the lowest part being the most prominent. A small quantity of fluid is indicated only by lateral bulging in the flanks. A large quantity not only bulges the flank, but rounds the anterior aspect of the belly, pushes outwards the lower ribs, and causes the whole abdomen to become barrel-shaped; the umbilicus is frequently stretched, and the whole surface exceedingly tense.

3. To meteorism, *i.e.*, distension of the bowel by gas. Great enlargement of the abdomen, caused by excessive meteorism, is often a conspicuous feature in cases of stricture involving the rectum or sigmoid flexure.

4. Rarely to gas in the peritoneal cavity. In the two last conditions a change in the posture of the patient does not affect the shape of the abdomen.

5. To great enlargement of the uterus, ovary, spleen, bladder, or other organ; or to a retro-peritoneal myxo-sarcoma.

Unsymmetrical Enlargements are caused :—

1. By distension of the stomach or of a portion of the bowel; or by an increase in size of any of the other viscera.

2. By tumours of the various organs, or of other structures within the abdomen, or in its parietes.

Depression of the diaphragm, too, from intra-thoracic disease tends to protrude the abdominal wall; thus a general fulness may be observed in emphysema, a local fulness below the costal arch when there is effusion into the pleura on the same side.

Retraction.—A diminution in volume of the abdomen is observed as a sign of general emaciation; also in lead poisoning, and in stricture of the œsophagus or at the cardiac end of the stomach. In children suffering from basilar meningitis a boat-shaped hollow is frequently noticeable.

Movements.—In addition to the movements of respiration, the abdominal parietes may be affected by the peristaltic movements of the stomach and intestines, by pulsatory movements of the heart and aorta, or by the movements of the fœtus in the gravid uterus.

The **Respiratory Movements** are **increased** when those of the thorax are restricted, in emphysema, pleuritic effusion, and extensive consolidation of the lung.

They are **diminished** in pericarditis and diaphragmatic pleurisy; in peritonitis, or other painful affections of the abdominal organs. When the diaphragm is paralysed, the hypochondriac regions sink in when a deep inspiration is taken, but no other abdominal movement is observed. (See p. 138.)

Peristaltic Movements are but rarely visible in health. They indicate distension of the stomach or intestines, together with in many cases hypertrophy of their walls. Thus in stricture of the pylorus, when the stomach becomes dilated and its walls thickened, the greater curvature is often visible at a lower level than the navel, and peristaltic waves may be seen passing from left to right, and occasionally even in the opposite direction. In cases of narrowing or occlusion of the intestine, the extent of the peristaltic movements may furnish useful information as to the seat of the lesion.

Pulsating Movements may be due to aneurysm of the abdominal aorta, or to tumours lying over this vessel; but more frequently abdominal pulsation occurs in anæmic or nervous subjects. Epigastric pulsation has been already mentioned (see p. 201). Occasionally in great ascites the heart's impulse may be visible as a superficial wave passing over the surface of the abdomen.

The Umbilicus, lower in children than in adults, becomes everted, or even obliterated in ascites, pregnancy, or other conditions which distend the abdomen. Distension of the upper part is apt to lower the position of the umbilicus, distension of the lower part to raise its position. In œdema of the abdominal parietes it is often sunk below the surface. It may be protruded by a hernia or its tissues infiltrated and thickened by a new growth; thus it is sometimes concomitantly affected by cancer in cases of cancer of the stomach or omentum. A fæcal fistula may form at the umbilicus in connection with tubercular disease of the intestine; in rare cases it is dependent on a patent Meckel's diverticulum.

A **Dilatation of the Superficial Veins** points to engorgement of the portal system, which may be caused by disease of the terminal branches of the portal vein, as in alcoholic cirrhosis of the liver; by disease or pressure on the trunk of this vein or the inferior vena cava. In the last case the veins of the lower limb become enlarged, and there is generally some œdema. The superficial veins of the lower part of the abdomen are frequently very conspicuous in cases of chronic dilatation of the stomach.

The Skin is often pigmented during pregnancy, especially around the navel and lower part of the linea alba; and still more markedly in Addison's disease; also in other forms of chronic abdominal disease, as tubercle, cancer and malaria. White or bluish lines (*lineæ albi-*

cantes), together with a bluish-white colour of the general surface, are met with as a result of great or repeated distension of the abdomen.

Palpation of the Abdomen.—In the investigation of abdominal disease, palpation is by far the most valuable method of examination. To carry it out efficiently the abdominal muscles should be as relaxed as possible, and this is best attained as follows :—

(1.) The patient should lie on his back with his shoulders well raised and supported, the chin touching the sternum and the knees drawn up.

(2.) The examiner's hands, previously warmed, should be placed gently on the abdomen, and then pressed in evenly and firmly while the patient is engaged in conversation, or after he has made a forcible expiration. In this way the abdominal cavity may be pretty thoroughly explored.

(3.) Occasionally, owing to unyielding tension of the recti muscles, the administration of chloroform is required.

Normally the surface of the abdomen, while generally soft and yielding to pressure, is somewhat firm over the recti muscles, and in the epigastrium over the liver. By deep pressure the kidneys, aorta, and even the vertebral column may frequently be felt.

Pain on Pressure.—This occurs in all inflammatory conditions affecting the walls or contents of the abdomen. General tenderness is usually due to inflammation of the peritoneum, but may occur in hysterical subjects, when there will probably be hyperæsthesia in other parts. Localised tenderness is significant of an affection of some underlying structure; but it may result from irritation of any portion of the sensory tract between the skin and the spinal cord, as from herpes zoster, pleurisy, or pachymeningitis. Tenderness in the epigastrium occurs in cases of abdominal aneurysm, in incipient caries of the spine and in gastric ulcer.

Resistance.—As a rule, the sensation of increased resistance to palpation is an indication that some morbid change exists beneath the part palpated; its presence and position should be carefully observed, for it may be the only palpable sign of a deep-seated lesion. But hard tense muscles are met with in hysterical or neurotic subjects apart from internal disease. Frequently, too, they are associated with distinct swelling, caused partly by local distension of the intestine, and partly by muscular contraction. Sometimes a dull note is obtained over the prominence, and the resemblance to a new growth may be great. But these "phantom tumours" come and go, and usually disappear when chloroform is administered, or when gas is removed by means of a tube passed into the rectum. The abdominal wall becomes hard, knotty and retracted in ordinary colic, whereas in lead colic it is often soft and supple.

from others arising within the abdominal cavity; the former are more easily felt when the patient is on his hands and knees, and often become more apparent when the patient contracts the abdominal muscles in their neighbourhood.

Lastly, the possibility that an abdominal swelling is merely a fæcal tumour should always be considered, and hence it is highly advisable to thoroughly empty the bowels before arriving at a positive conclusion with regard to any abdominal tumour. (*See* Intestine.)

Percussion.—The knowledge derived from percussion with regard to the size of the solid viscera and the state of distension of the stomach and bowels is referred to under the examination of the individual organs. In the present place it is only necessary to consider percussion in relation to the condition of the peritoneal sac.

Liquid in the Sac.—The smallest effusion accessible to percussion is one which rises a little above the level of the pelvic cavity; this yields a dull note over the lowest part of the abdomen when the patient's body is erect, the dulness disappearing when the patient lies down. A larger effusion produces dulness in the flanks when the patient is on his back, and also in the hypogastric region. If the effusion be free, the site and extent of dulness vary with the position of the patient; thus, if he turn on one side, the level of dulness rises on the dependent side, while the dulness of the uppermost flank gradually diminishes or disappears altogether, being replaced by clear or tympanitic resonance.

A very great effusion may in exceptional cases render the whole surface of the abdomen dull to percussion, but, as a rule, a small area of resonance still remains in front above the umbilicus. In all cases of effusion, too, a tract of clear resonance may usually be detected on each side between the axillary and scapular lines, that is in situations which correspond to the positions of the ascending and descending colon.

It is, however, a matter of frequent observation that considerable ascites may exist without any dulness; when this is the case, the diagnosis is based partly on the size of the abdomen and on the results of palpation, and partly on a consideration of the general aspects of the case.

It is also to be noticed that dulness due to the presence of a liquid effusion may not alter its position with that of the patient, but remain fixed to one flank or to some other part of the abdomen; this is usually, but not always, to be explained by the presence of adhesions which shut off the liquid from the rest of the peritoneal cavity.

Gas in the Sac.—A large quantity of gas in the peritoneal cavity yields a uniform tympanitic sound to percussion over the whole abdomen, and this resonance encroaches upon or replaces the special sounds of the liver, stomach, and spleen.

Conditions simulating Ascites.—The most common are (1) large

cysts—ovarian, renal, or hydatid; (2) a distended urinary bladder; (3) a pregnant uterus.

An **Ovarian Cyst** is distinguished by the following points:—

(1.) There is commonly a history of a swelling beginning in one iliac region.

(2.) The greatest girth of the abdomen is usually below the umbilicus, whereas in ascites it is at the level of or above the umbilicus.

(3.) The umbilicus is at a higher level than normal, but in ascites it is commonly, as in health, about an inch nearer the pubis than the sternum.

(4.) There is dulness in front and resonance in the flanks, the reverse being usually the case in ascites.

(5.) The fluid obtained by tapping an ovarian cyst is often glutinous, or of brownish colour from admixture with blood.

(6.) Vaginal examination is also of assistance in diagnosis.

A **Renal** or a **Hydatid Cyst** is usually easily distinguished from ascites by the unilateral origin of the swelling, by the nature of the fluid, and by the history and other general features of the case.

Causes and Differential Diagnosis of Ascites.—The chief causes of ascites, together with some of the leading indications upon which the diagnosis of a particular case is based, are arranged from a clinical standpoint in the following table:—

I. Ascites is **not associated with dropsy** elsewhere.

1. Abdominal pain and tenderness are severe; the abdomen is usually much enlarged from distension of the intestines, but the quantity of ascitic fluid is usually small, and insufficient to give distinct fluctuation. Vomiting, constipation, pyrexia and collapse are other marked features of the case. *Acute Peritonitis.*

2. Abdominal pain and tenderness are moderate or slight.

(*a.*) Tumours of various sizes, shapes and consistence may be felt.

α. The abdomen is retracted or moderately enlarged. The patient is usually young, is emaciated, subject to night sweats, and to irregular action of the bowels. There may be signs of tubercle in the lungs, or in other organs. Frequently there is redness and œdema about the umbilicus. *Tubercular Peritonitis.*

β. The abdomen is greatly enlarged; there is vomiting and emaciation, and the patient is usually past middle life.

(1.) Fluctuation is distinct, and nodules may be felt near the umbilicus; there may be evidence of cancer of the stomach, ovary, or other organ. *Cancer of Peritoneum.*

(2.) Fluctuation is indistinct; hard, irregular, or gelatinous masses may be felt; the fluid obtained by tapping is viscid or thin and turbid, with blood and colloid cells. *Colloid Disease of Peritoneum.*

(*b.*) No tumours to be felt; the abdomen is moderately enlarged, and constitutional disturbance is usually moderate in degree. *Chronic Peritonitis.*

3. Abdominal pain and tenderness slight, absent, or limited to hepatic region.
   (*a.*) There is much ascites; the superficial veins are distended; there are venous stigmata on the cheeks; the liver is sometimes palpably enlarged, sometimes not, and its dulness may be diminished; the spleen is often enlarged.
   Cirrhosis of the Liver.
   (*b.*) Degree of ascites variable; superficial veins not usually enlarged; jaundice commonly present; irregular or nodular enlargement of liver; spleen not enlarged. *Cancer of Liver.*

II. Ascites is **associated with dropsy** elsewhere.
1. Dyspnœa and œdema of legs, before and out of proportion to degree of ascites.
   (1.) Physical signs of dilatation of right ventricle. *Tricuspid incompetence from mitral disease, or from bronchitis and emphysema, or from weakness of cardiac muscle.*
   (2.) Rapidly progressing œdema, great enlargement of superficial veins of chest and abdomen, and perhaps physical signs of a tumour or indications of pressure upon œsophagus or lungs.
   Tumour compressing Inferior Vena Cava.
2. Œdema beginning in the face, and often associated with hydrothorax, albuminuria, and other signs of renal disease. *Kidney Disease.*

THE STOMACH.

Anatomy.—Five-sixths of the stomach lie to the left, one-sixth to the right of the mesial plane of the body. The cardiac orifice, placed deeply, lies in front and to the left of the eleventh dorsal vertebra; opposite to it anteriorly is the sternal end of the seventh costal cartilage. From the cardia the small curvature runs at first vertically downwards along the left side of the vertebral column, and then bends across at about the height of the first lumbar vertebra to end in the pylorus, which is situated a little to the right of the middle line, at the level of the tip of the ensiform cartilage, its right border corresponding to the union of the seventh and eighth costal cartilages. The small curvature is completely covered by the left lobe, the pylorus by the right lobe of the liver.

The fundus, the highest part of the stomach, rises into the left hypochondrium, and is partly covered by the left lung. The great curvature, descending from behind the left lung, passes through the left hypochondrium and epigastrium to reach the pyloric end near the gall bladder. The position of the great curvature varies greatly according to the state of distension of the stomach; an average position in the middle line is about midway between the ensiform cartilage and the umbilicus, and in health it is quite exceptional for the great curvature to extend as low as the umbilicus.

Symptoms present in Stomach Disorders.—A person suffering

from a disorder of the stomach usually complains of some symptom which is related to the taking of food, such as pain or fulness after meals, flatulence, acidity, heartburn, nausea, or vomiting. But the chief complaint may be of the heart; the patient is conscious of its movements, or suffers from palpitation, and sometimes indeed believes that there is actual disease of the heart. Occasionally, and especially when there is malignant disease of the stomach, the patient lays the greatest stress on loss of flesh and strength, whilst it is to be noticed that in all stomach affections there is a tendency to mental anxiety and depression.

Common to all diseases of the stomach is the presence of pain over the seat of the organ, that is, the epigastrium. In association with this pain the patient may complain of pain in other situations, as over the front of the chest below mid-sternum; between the shoulders at the level of the fourth and fifth dorsal vertebræ; while sometimes there is a feeling of constriction and oppression in the left side. In addition to these localities the head is often the seat of aching pains; sometimes there is occipital headache, but generally the patient suffers from severe pain across the forehead.

The abdominal pain of gastric ulcer occurs some time (a quarter to two hours) after a meal, that of gastric catarrh immediately after food—the former is usually situated at a higher level than the latter, which often corresponds to the position of the lower curvature, and may be limited to the back in the neighbourhood of the eleventh and twelfth dorsal vertebræ. The pain of gastric ulcer may abate when the patient lies in bed, but that of catarrh or cancer tends to be persistent.

The early symptoms of cancer in the stomach frequently comprise a sensation of fulness and discomfort after food, together with a more or less constant feeling of uneasiness in the epigastric region.

Inspection and Palpation.—Dilatation of the stomach gives rise to a uniform somewhat oval prominence in the epigastric and left hypochondriac regions, and the outline of the greater curvature may be distinctly seen. If the dilatation is excessive the whole abdomen becomes enlarged, and even tensely prominent. Frequently in chronic dilatation the stomach occupies a lower position than normal, and then the epigastrium may be flat or depressed, instead of rounded. Sometimes in such cases peristaltic movements are so marked that the whole outline of the stomach becomes distinctly visible.

Artificial Inflation of the stomach by carbonic acid gas is sometimes of much value in diagnosis; for by this method we are enabled to form an opinion with regard to the shape and size of the stomach, the capabilities of the pyloric orifice, together with the relative position

and immediate connections of any tumour. It is easily performed by giving to the patient first one or two teaspoonfuls of tartaric acid dissolved in water, and then, after an interval of a few seconds, the same quantity of a solution of bicarbonate of soda. In a short time the epigastrium bulges out, and the greater curvature may be perceptible as low as, or more commonly a little above, the level of the umbilicus. If any untoward symptoms arise, such as gasping for breath, or much general distress, they may be promptly relieved by the introduction of the stomach tube. Sometimes, owing to ulceration or cancerous deposit, the pylorus is more or less incompetent; then the carbonic acid gas will pass directly into the duodenum, and consequently inflation of the stomach cannot be satisfactorily brought about.

Pain on Pressure.—While periodic diffuse pain and a varying degree of general tenderness are common to all affections of the stomach, both may be entirely absent even when the viscus is the seat of well-marked disease.

A localised pain in the epigastrium, or behind over the lower dorsal and upper lumbar vertebræ, which is usually aggravated by pressure, is significant of gastric ulcer. There may be tenderness, too, in the neighbourhood of a cancerous tumour. Tenderness in the epigastrium, or at a lower level, is met with in gastric catarrh, whereas in cases of atonic dyspepsia pressure with the hand often gives the patient relief.

Increased Resistance in the epigastric or umbilical region, and especially over the upper portion of the right rectus muscle, may be the only palpable evidence of a tumour of the stomach. Tenseness of the epigastrium also occurs in cases of ulcer, and in gastritis; while when the stomach is much dilated from any cause, and its walls are hypertrophied, there is increased resistance to pressure often over an extensive area.

A Tumour above and a little to the right of the umbilicus is suggestive of scirrhus of the pylorus, for this part is easily dragged down from its normal position at the right costal margin. The mass is usually circumscribed, except on the left, where sometimes it may be felt to shade off gradually into the wall of the stomach. Its size is usually less than that of a Tangerine orange. It is unaffected by the movements of respiration, although occasionally a slight descent may be felt during a deep respiration, and especially if the stomach tumour is connected with the liver or adherent to the diaphragm.

In a suspected case of cancer of the pylorus, when there is no obvious prominence to be seen or felt, there is one place especially which requires the most careful investigation; this is the edge of the right rectus muscle above the level of the umbilicus. The observer, standing

on the right side of the patient, should push the tips of his fingers as deeply as possible beneath this edge, while the patient lies on his back, turns on one or other side, or leans forwards. In this way a small nodule may be discovered which otherwise would have escaped notice.

It is also to be observed:—(1.) That a tumour may displace the pylorus to a considerable extent; thus cancer of this part is often situated to the left of the middle line, occasionally even as far out as the left hypochondrium; and in rare cases a tumour has been felt below the level of the umbilicus. (2.) That the pulsation of the aorta may be transmitted through the tumour. (3.) That such pulsation, and even the tumour itself, may sometimes disappear for a few days.

A tumour in the epigastrium and left hypochondrium occurs in cancer of the body of the stomach; and ridges may be felt when the curvatures are markedly involved. In these regions, too, a firm, smooth hard mass is perceptible in such rare conditions as diffuse sarcoma, or chronic induration of the wall of the stomach; evidence of the latter condition is usually most distinct to the right of the middle line, where also induration around an ulcer may sometimes be felt.

Tumours involving the cardiac orifice or the lesser curvature are not easily reached by the fingers. Cancer at the cardiac end of the stomach is indeed very difficult to diagnose; no tumour may be felt, and hæmatemesis is usually absent; there is commonly great anæmia, and the case often resembles one of pernicious anæmia, but the age is more advanced, and the skin and retina are free from hæmorrhages.

Very rarely hard irregular masses in the epigastrium are due to gastric concretions, such as hair, string, &c.

Fluctuation and **Splashing Sounds** are commonly obtained in cases of great gastric dilatation by palpating the lower part of the abdomen.

Percussion.—The outline of the stomach cannot, in normal conditions, be mapped out by percussion, for only the great curvature and a small portion of the anterior surface are in direct contact with the abdominal parietes. Over this superficial portion a clear low-pitched tympanitic note is commonly obtained, which may suffice to separate the stomach from the liver above, the lung and spleen to the left, and the colon below. But the character of the percussion note will obviously vary greatly at different times in relation to the contents of the stomach and the tension of its walls; solids and liquids producing a muffled or dull note, great distension from gas imparting a metallic quality over a larger or smaller area. The note over the transverse colon is commonly higher in pitch than that over the stomach, but if the latter contain but little gas, while the colon is distended with it, it will probably be impossible by means of percussion to define the greater curvature which forms the line of separation between the two

organs. The following methods are sometimes of service when it is considered necessary to form a true estimate of the size of the stomach, or to determine the position of the great curvature.

1. Artificial inflation of the stomach with carbonic acid gas, as already described on page 264.

2. The stomach being empty, the abdomen is percussed while the patient's body is erect; under these conditions the boundaries of the stomach will be indistinct. But if now the patient drink a large quantity of water—the erect posture being maintained—a dull area will be found crossing the linea alba, the lower boundary of which may be taken as the line of the greater curvature.

The second is a much less satisfactory method than the first.

Traube's Semilunar Space.—This is that portion of the left hypochondrium in which the fundus of the stomach lies uncovered. It extends vertically from the sixth to the ninth cartilages, and transversely from the heart's apex beat to the anterior axillary line. The chord of the semicircle is formed by the left costal margin, and the arc, only slightly convex, is bounded by the apex of the heart, the left lung, and the spleen.

Diminution of the Gastric Tympanitic Area is produced by enlargement of the spleen or of the left lobe of the liver, by pericardial effusion, by hypertrophy of the heart, or by fluid in the cavity of the left pleura.

In the last condition, dulness in the upper part of Traube's space is an early phenomenon, and as the effusion increases the space becomes smaller, till finally in some cases it is completely obliterated, dulness being obtained as low as the costal margin. In pneumonia of the lower lobe of the left lung, Traube's space is rarely encroached on; hence some Continental writers have regarded a distinct diminution in the area of the semilunar space as a point in favour of pleurisy, but in our experience a tympanitic note ascending high into the axilla in cases of pleuritic effusion is by no means of rare occurrence.

Enlargement of the Gastric Tympanitic Area occurs—

(1.) Most markedly in chronic dilatation of the stomach, which may be caused either by obstruction at the pyloric end, or by weakness of the wall of the stomach. Then a tympanitic note is obtained over a wide area, sometimes even as low as the pubes. Particular attention should be given to Traube's space, for minor degrees of gastric dilatation are often only indicated by an extension upwards and to the left of this tympanitic area. If the stomach contain liquid as well as gas, the percussion sound will vary with change of posture. Here may be mentioned the tympanitic note at the left base which is sometimes found below the dulness due to pleuritic effusion.

(2.) In the rare condition described by Fagge as acute paralytic distension; here the front of the abdomen is resonant, but there is usually dulness and fluctuation in the pubic region.

(3.) In downward dislocation of the stomach as from tight lacing, or from tumours which mechanically drag down the pylorus.

Tumours of the stomach, unless very massive, do not yield an absolutely dull sound, but commonly a muffled tympanitic note.

Auscultation.—This method is of little value in diagnosis. In healthy persons, shortly after taking much liquid, metallic or gurgling sounds may be heard on applying the stethoscope over the stomach, when, at the same time, sudden pressure is made over the upper part of the abdomen. Sometimes such noises heard in the neighbourhood of the left hypochondrium may closely resemble pulmonary metallic râles.

In contraction of the cardiac orifice, on auscultating the epigastrium, the noise of swallowing is much delayed, and heard as short metallic drops. In dilatation of the stomach, with fermentation of the contents, spontaneous bubbling râles from bursting of gas bubbles may be audible.

The **succussion splash** is also a valuable sign of this condition; it is obtained by shaking the patient while the ear is held over his stomach. If no fluid has been taken for four hours, the succussion sound may be taken as almost pathognomonic of gastric dilatation.

EXAMINATION OF VOMITED MATTERS.

Vomited materials consist of food-constituents in various stages of digestion, mixed with gastric juice, the secretions from the nose and mouth, and frequently also with bile. Other materials, as blood, may also be present.

Quantity.—This will obviously vary with the amount of food or liquid in the stomach at the time of vomiting, and with the intensity and duration of the act.

A small quantity of glairy mucus is common in gastric catarrh, the result of alcoholism, cirrhosis of the liver, or heart disease.

Very large vomits occur in dilatation of the stomach, whether due to enfeeblement of its walls or to stricture of the pylorus; and it is characteristic of this class of cases that excessive quantities of fluid, often several pints, are brought up at a time, and at intervals of two or three days.

Large quantities of a watery fluid are ejected in cholera; of blood in ulcer of the stomach.

Reaction.—The vomit is acid in acute and chronic gastric catarrh;

in dilatation of the stomach the acidity may be sufficiently intense to produce red spots on cloth.

The vomit is **alkaline** sometimes in chronic gastritis, and when there is much blood present. The watery fluid met with in pyrosis and in Asiatic cholera is usually neutral or alkaline.

Smell.—As a rule, vomited materials have a sour smell, which is variously modified by the different kinds of food taken.

The presence of fatty acids is recognised by their peculiar penetrating odour. A beery smell points to fermentation of the stomach contents. A fæcal odour occurs in cases of intestinal obstruction, and an ammoniacal one in cases of uræmia. A peculiarly repulsive cadaverous fœtor is often observed when the vomit contains much blood, and especially if it be derived from a cancerous ulcer

In cases of poisoning, the smell of the vomit may be of help in diagnosis; thus, in phosphorus poisoning a characteristic odour is noticed, in poisoning by nitro-benzol a smell of bitter almonds.

Consistence and General Appearance.—Food, more or less unaltered in character, is vomited in hysteria; in cancer of the cardiac end of the stomach; and in dyspepsia, whenever the secretion of gastric juice is imperfect either as regards quantity or quality. Partially digested food is rejected in cases of cancer of the pylorus and in many cases of dyspepsia. In acid dyspepsia much fatty matter is often found in the vomit, which occurs several hours after a meal. Certain articles of diet, such as coffee, cocoa, or red wine, colour the vomit so that it may look as if mixed with blood. A black coloured vomit occurs in cases of acute lead poisoning, and after the administration of preparations of iron or bismuth. Round worms are sometimes vomited by children; and less commonly thread worms, anchylostomæ or trichinæ, have been found in vomit. Membranous shreds of echinococcus should be sought when a hydatid tumour of the liver or spleen has burst into the stomach; under the microscope scolices and hooklets may be discovered. According to the predominance of certain constituents, vomits may be divided into the following categories:—

Watery Vomit.—(1.) "Pyrosis" or "water-brash" occurs in chronic catarrh, especially in that of drunkards; in ulcer and cancer of the stomach; and sometimes in "nervous dyspepsia." The fluid vomited is thin, watery, more or less clear, and consists largely of swallowed saliva, or is derived from glands at the lower end of the œsophagus; commonly neutral or alkaline, it may be strongly acid and secreted by the stomach. The specific gravity is 1004–1007. Sulphocyanide of potassium may be present, as indicated by a dark blood-red colour with ferric chloride. (2.) In cholera, a pale watery fluid of low sp. gr.

(1002–1007) is vomited in enormous quantities; whitish flakes float on the surface, which sink when the fluid is allowed to stand, and leave a yellowish or greyish liquid above; this usually contains urea, ammonium carbonate and sodium chloride, but is poor in albumen.

Mucous Vomit occurs in inflammatory conditions of the mucous membrane. The material is tough and gelatinous, sometimes colourless, sometimes stained green or yellow by bile, or streaked red with blood. Remnants of food are commonly present.

Bilious Vomit, oftener green than yellow, occurs whenever there is severe retching, and frequently accompanies cirrhosis of the liver.

A thick grass-green vomit is prominent in cases of perforative-peritonitis, or of severe bowel obstruction, and is also met with in cerebral affections. The flakes which float on its surface consist of pavement and cylindrical epithelium, of fat drops, and amorphous masses.

Purulent Vomit.—This is a rare condition, usually produced by the bursting of an abscess into the stomach; very rarely it is the result of suppurative inflammation of the walls of the stomach.

Stercoraceous Vomit.—True fæcal masses have only been observed in very exceptional cases. But greenish or yellowish vomit of disgusting fæcal odour is not uncommon in intestinal obstruction and in severe diffuse peritonitis. Such vomiting is of dangerous or fatal omen.

Hæmatemesis.—(1.) **Small Quantities of Blood.**—Whenever vomiting is accompanied by severe straining, florid streaks of blood are common, and especially so in congestion of the gastric mucous membrane, as from heart or liver disease.

When blood remains some time in the stomach, it is altered by the gastric juice—the red corpuscles and hæmoglobin being broken up and replaced by hæmatin—and becomes chocolate-coloured, or like "coffee grounds." The condition is frequent in cancer of the stomach, but occurs less commonly in ulcer and congestion of the stomach, unless the latter result from swallowing a strong acid.

Pure unchanged blood is very rarely vomited, and is scarcely ever so bright coloured as that derived from the lungs.

(2.) **Large Hæmorrhages**, up to several pints, are especially characteristic of gastric ulcer; the blood is usually darker and less frothy than that brought up from the lungs. Hæmorrhage sometimes occurs in cancer of the stomach, or as a result of portal congestion; it occasionally may arise from the opening of an aneurysm into the œsophagus or stomach, or in connection with the hæmorrhagic diathesis.

When blood is vomited, its source is not necessarily the stomach; it may be swallowed blood which has come from the nose, throat, or even from the lungs. In cirrhosis of the liver, the veins at the lower end of

the œsophagus are frequently enlarged and varicose, and blood derived from them may run down into the stomach and then be vomited.

It has been already mentioned that a red or black vomit may derive its colour from other substances than blood; it is therefore essential in all cases of dark vomits to apply one of the following tests:—

Tests.—1. Filter some of the "coffee ground" looking material; evaporate to dryness a small quantity of the filtrate, powder the residue and place it with a crystal of common salt on a glass slide, and lay a cover-glass on the preparation. Then let a few drops of glacial acetic acid flow beneath the cover-glass, and gently heat for some time, when brown or red crystals of hæmin will be seen under a high power of the microscope.

2. Treat a portion of the vomit with caustic potash, filter, and examine the filtrate with the spectroscope for the spectrum of hæmatin in alkaline solutions.

The **Microscopical Examination** of vomited matter is best carried out by decanting off the liquid part into a conical glass, and subsequently taking up a drop of the sediment with a pipette. The following are the chief constituents that may be present:—

1. **Food Particles.**

(*a.*) Striated muscular fibres, either intact or divided into a number of discs, or represented by a crumbling mass according to the stage of digestion. (*b.*) Involuntary muscular fibres. (*c.*) Elastic fibres and connective tissue. (*d.*) Fatty globules and fat needles; these are highly refractive, and dissolve in ether. (*e.*) Starch granules, which are recognised by their concentric arrangement, and by the blue stain they take with iodine.

2. **Epithelium.**—Flattened cells from the mouth and columnar cells from the stomach may be visible, together possibly with casts of the gastric follicles. Particles, too, of mucous membrane may be seen, perhaps stained with blood, and presenting the openings of gastric tubules.

3. **Red Blood Corpuscles** are seen unaltered only when a sudden hæmorrhage into the stomach has been immediately vomited; as a rule the corpuscles are represented by colourless rings.

4. **Leucocytes**, mucous, or pus corpuscles may also be present.

5 **Cancer Cells** are usually difficult to recognise.

6. **Fungi.**—(1.) **The Yeasts.**—(*a.*) The yeast plant or torula cerevisiæ consists of oval highly refracting cells, which are usually in groups of three or more, and often form branching chains. A few isolated cells are very common; when numerous, they are significant of fermentation having taken place in the stomach, and of abnormal starch digestion. Yeast cells are often seen in cases of chronic catarrh,

ulcer, cancer and dilatation of the stomach. They stain brownish yellow with iodo-potassic iodide solution. (*b.*) *Thrush* fungus very rarely may be detected in the vomit.

(2.) **Schizomycetes.**—Many varieties of micrococci, bacteria and bacilli are met with in vomit. Of micrococci the **sarcina ventriculi** is frequently present, along with yeast cells, in gastric dilatation. These cocci are commonly in square groups of four or some multiple of four. They resemble wool packs, have a dark silver-grey tint, and turn a mahogany-brown or a reddish violet colour on adding a drop of iodo-potassic iodide solution. They are rendered more distinct by the addition of liq. potassæ.

(3.) **Moulds.**—Filaments and spores are occasionally seen, but are of no known pathological significance.

INVESTIGATION OF THE CONTENTS OF THE STOMACH AND OF ITS ACTIVITY DURING DIGESTION.

To make a complete examination of any case of stomach disorder, it is necessary to investigate the process of digestion by every means at our disposal, in order to obtain information with regard to—

1. The duration of gastric digestion.
2. The condition of the contents of the stomach during digestion.
3. The motor power of the stomach during digestion.
4. The absorptive power of the stomach during digestion.

And although in practice it is not always necessary or even desirable to withdraw the contents of the stomach, there are cases in which it is impossible to make an accurate diagnosis, and to administer other than empirical treatment, without first submitting the gastric contents to careful and repeated examination.

1. The Duration of Gastric Digestion varies with the kind and quantity of food taken, and to some extent in different persons. To determine the duration in a particular case, it is necessary to give a standard meal, consisting of some clear soup, a chop or steak, and a piece of white bread, and to forbid any other food to be taken for the next six or seven hours; after this interval the stomach is to be emptied.

Method.—To empty the stomach a soft gum tube, having numerous fine perforations in its end, should be introduced into its cavity until a slight hindrance is encountered. If the viscus is moderately full its contents flow out at once, but if it is nearly empty, it may be necessary to press sharply over the epigastrium, or to pinch the tube with one hand, while the fingers of the other hand squeeze out the air from the free portion of the tube; a vacuum is thus created, and any liquid in

the stomach will at once ascend the tube. Sometimes, however, it is necessary to rinse out the stomach with a little warm water in order to get any gastric juice. This is done by attaching a glass funnel to the tube, raising both above the level of the patient's head, and pouring warm water into the funnel; then, before the funnel is quite empty, it should be quickly lowered over a vessel which receives the gastric contents. The tube must be introduced with great care when the presence of a simple or cancerous ulcer is suspected, and must not be passed at all when there is a tendency to hæmatemesis, or when, owing to the taking of some corrosive poison, a severe destruction of the wall is likely to be present.

If at the end of seven hours the rinsings of the stomach contain nothing more than a few flakes or shreds of food remains, it is probable that digestion is normal, or at least it may be affirmed that there is no obstacle to the exit of food from the stomach, and that the gastric secretion is sufficiently active. The gastric juice may, however, be in excess of normal, or unduly active; then the period of digestion would be shortened. To ascertain if this be so, the tube should be introduced at an earlier period, say five hours after the standard meal was taken, and thus at length a conclusion may be come to with regard to the exact duration of digestion.

But if after seven hours there are considerable remains of food in the stomach, digestion is retarded either (1) because the secretion is inactive, or (2) because there is a hindrance to the escape of the products of digestion in consequence of pyloric stenosis, diminished peristaltic action, or weakness of the gastric walls.

2. The Condition of the Gastric Contents or the Chemistry of Digestion.—In health, after the ingestion of food the acidity of the gastric juice is due at first to the presence of lactic acid, but later mainly to hydrochloric acid, which usually attains its maximum quantity about one hour before the completion of digestion. It is at that period that we should endeavour to obtain a specimen of gastric juice. Instead of the standard meal already mentioned, it is more convenient to provide one which is digested in a shorter time than seven hours; thus if a patient take one cup of weak tea and a little dry toast, a suitable specimen of gastric juice can be obtained after an interval of one hour. [It must not be forgotten, too, that if the patient has vomited, the filtrate of the vomit may often be taken as a reliable specimen of the gastric secretion, although it is frequently necessary to obtain a purer specimen.]

Examination of the withdrawn contents.—1. Examine (just as in the case of vomited matters) for bile, blood, mucus and pus. Then filter.

2. The solids on the filter are first to be inspected, in order to ascertain the degree of maceration, and then to be examined microscopically. (See under *Vomit.*)

3. Examination of the Filtrate—Reaction. If acid to litmus paper, the acidity may be caused by one or more of the following substances :—

Free hydrochloric acid.
Free lactic ,,
Free butyric ,,
Free acetic ,,
Acid phosphates.

To determine whether the acidity be due to the presence of acid phosphates or to the presence of one of the free acids, add a few drops of *Congo red* to a portion of the filtrate. In the former case the reagent is unaltered, in the latter case it turns blue.

A Blue Reaction is obtained with Congo Red.—Butyric and acetic acids are sufficiently recognised by the smell of rancid butter and vinegar respectively.

Tests for hydrochloric acid.—(1.) Phloroglucin-vanillin. This reagent is made by dissolving two parts of phloroglucin and one part of vanillin in thirty parts of absolute alcohol. A few drops of the filtrate are slowly evaporated to dryness in a porcelain dish, then a drop of the reagent is allowed to run over the dried residue; if hydrochloric acid be present, a delicate rose-red tinge quickly appears. The test is delicate enough to detect 0.06 per cent. of the acid, and the reaction is not hindered by the presence of proteids.

(2.) Methyl violet. A weak watery solution of this reagent is decolorised by much hydrochloric acid, but a small quantity of hydrochloric acid changes the violet into a blue colour.

(3.) Tropæolin (oo).—An alkaline solution of tropæolin is changed from yellow to a ruby or brown-red colour, by a trace of hydrochloric acid or of lactic acid.

Tests for lactic acid.—(1.) Ferric carbolic solution. This is made by mixing 10 ctm. of a 4 per cent. solution of carbolic acid with 20 ctm. of distilled water, and then adding one drop of liquor ferri perchloridi. The amethyst blue colour of this solution is turned yellow or yellowish-green by lactic acid; it becomes colourless if hydrochloric acid only is present. By this method 0.01 per thousand of lactic acid can be detected. Alcohol, sugar and phosphates, however, give a straw-yellow colour with the iron carbolic solution; and if this rather than a greenish-yellow colour is obtained, it will be necessary to shake up some of the filtrate with ether in a test-tube; the ether should then be poured off into a dish and evaporated over hot water without a flame.

The residue is then dissolved in a little water and tested for lactic acid with the iron carbolic reagent.

(2.) The faint yellow colour of a very dilute solution of perchloride of iron becomes intensified by the addition of a little dilute lactic acid, but is unaffected by dilute hydrochloric, butyric, or acetic acid.

The gastric juice shows little or no hydrochloric acid in many cases of cancer of the stomach, in acute febrile diseases, and in certain forms of dyspepsia. An excess of hydrochloric acid is common in cases of gastric ulcer, and in some varieties of acid dyspepsia, while in other varieties the acidity is due to an excess of lactic acid.

3. The Motor Power of the Stomach is ascertained by giving the patient salol, and then testing the urine every half hour for the presence of salicylic acid by dropping in ferric chloride solution. The purplish colour should appear in some 50 or 60 minutes. If it cannot be obtained after $1\frac{1}{2}$ to 2 hours, then in all probability the viscus is dilated.

4. The Absorptive Power of the Stomach is roughly determined by the administration of iodide of potassium (3 grs. in capsules), and then testing the saliva every ten minutes.

When there is normal absorption from the stomach, a reaction is obtained in about fifteen minutes.

THE INTESTINES.

Anatomy.—The following are some of the chief points in the medical anatomy of the intestines :—

Duodenum.—The first part is invested by peritoneum like the stomach, but the second and third parts are covered by peritoneum only in front ; behind, they are in close relation with connective tissue. The first part of the duodenum is movable, while the end of the third part where it joins the jejunum is firmly fixed by a fibrous band, and remains in the same position when the rest of the duodenum, in consequence of dilatation, may form a loop hanging down towards the iliac crest. The duodenum is surrounded by many important organs, and may be implicated by disease affecting them. Thus above and in front of the first part are the liver and gall bladder, behind it the bile duct and portal vein. The second part embraces the head of the pancreas, and receives the bile and pancreatic ducts. The third part runs obliquely upwards from right to left in front of the aorta and vena cava, at the level of the second lumbar vertebra ; in front of it are the superior mesenteric vessels.

Meckel's Diverticulum.—Not very rarely a pouch or diverticulum, representing the remains of the vitelline duct, is given off from the

lower part of the ileum. It may reach as far as the umbilicus, but more commonly it is only a few inches in length. It is of importance in connection with intestinal obstruction.

The Cæcum, or that part of the colon which is below the entrance of the ileum, is usually situated in the right iliac fossa, with its apex pointing towards the middle of Poupart's ligament.

The Vermiform Appendix varies in length and in direction. As a rule it is about four inches long, and lies behind the end of the ileum, being directed upwards and to the left, but often enough it is found lying behind the cæcum, and then the results of its inflammation may be masked by the resonance of the cæcum in front. Sometimes the appendix projects downward towards the pelvis. In consequence of the varying length and direction of the appendix, and partly too in consequence of the tendency of a diseased appendix to form adhesions with neighbouring organs, an abscess in connection with it may occupy various situations, as the pelvis or the iliac fossa; it may project into the inguinal canal, or beneath Poupart's ligament; in other cases pus may burrow upwards towards the umbilicus, or it may surround the right kidney and form a large perinephritic abscess.

The Large Intestine.—The hepatic flexure lies under the liver, and is in intimate relation with the gall-bladder; the splenic flexure is behind the stomach; while the transverse colon crosses the abdomen, so that its lower border reaches nearly as low as the umbilicus. The sigmoid flexure, the narrowest part of the colon, occupies the left iliac fossa; it is freely movable, and its situation varies with its condition as to emptiness or distension. The rectum in the adult is situated entirely within the true pelvis. In the child it is also partly in the abdominal cavity, and is straighter and more vertical than in the adult.

Apart from indications of general disturbance, such as the condition of the temperature, of the strength and nutrition of the body, the important **symptoms** to be considered in connection with intestinal disease are vomiting, pain and disorders of defæcation (see Chap. II.), together with the results of a physical examination of the abdomen.

Inspection and Palpation.—**Tenderness** more or less general in distribution occurs in catarrh or ulceration of the bowels. Tenderness limited to the right iliac region is common in enteric fever, in typhlitis and in tubercular disease; when present in the left lumbar and iliac regions, catarrh of the descending colon, simple or dysenteric, is indicated.

Swellings in Relation with the Bowel.—A portion of the intestine

may be found in a state of distension, or it may be the seat of a palpable tumour.

Distension.—Flatulent distension of the intestines gives rise to general or local enlargement of the abdomen. The former is seen in cases of acute peritonitis where the whole of the bowel becomes distended, the latter when there is obstruction of a part of the bowel. In obstruction at the cæcum, the flanks may be flattened when the rest of the abdomen is much enlarged. In stricture of the sigmoid flexure the colon becomes enlarged, and peristaltic movements are often distinct, owing to hypertrophy of its walls; in such cases, that is when the obstruction is situated in the terminal portion of the colon, there is a great tendency for the symptoms to be referred back to the cæcum.

A Palpable Tumour is commonly produced by one of four causes, viz., fæcal accumulation, inflammatory exudation, new growths, intussusception.

1. **Fæcal Accumulation.**—Cylindrical fæcal masses are often palpable in the course of the colon, especially in the neighbourhood of the flexures, and they are particularly common down the left side of the abdomen. They may be found, however, in almost any part of the abdomen, in consequence of the strange positions the colon is liable to assume. Thus a loaded cæcum may be considerably displaced upwards or towards the left side, a loaded sigmoid flexure may form a tumour on the right side of the abdomen; similarly, inspissated fæces in the transverse colon may be felt below the umbilicus. A fæcal tumour is usually cylindrical in shape, has an uneven surface, and feels as if it were situated immediately beneath the abdominal wall; its consistence varies from softness to almost stony hardness. It may here be observed that liquid fæces in a dilated cæcum and ascending colon may be mistaken for fluid in the peritoneal cavity. Fæcal tumours often simulate cancerous and other masses; hence, in doubtful cases, it is important to clear out the bowels by purgatives and enemata.

2. **Inflammatory Swellings** are most commonly situated in the right or left iliac fossa. A swelling in the right iliac fossa is usually due to typhlitis or perityphlitis. Evidence of these affections is first afforded by tenderness, slight fulness and increased resistance to palpation; then a swelling forms, which may be doughy or firm in consistence; it occupies the space between the anterior superior spine of the ilium and the pubes, being usually at a little distance above Poupart's ligament, and may extend up into the lumbar region. It is associated with pain, tenderness, and symptoms of febrile disturbance. An abscess may form, and point externally; or it may burrow into the connective tissue of the pelvis, or upwards towards the kidney or umbilicus; or it may

burst into the intestine or into the peritoneal cavity. In some cases the abscess is situated behind the cæcum, and an exploratory puncture may be necessary to clear up the diagnosis. It must not be forgotten that an abscess in the right iliac region may result not only from inflammation of the appendix but also from inflammation of neighbouring or distant parts; thus it is met with as a result of caries of the spine, or of disease of the kidney or uterine appendages.

In the left iliac region an elongated, more or less firm swelling sometimes occurs in connection with dysentery or dysenteric diarrhœa.

3. **Cancer** of the cæcum produces a harder and more nodular mass than that due to inflammation about the cæcum, and the tumour is commonly nearer Poupart's ligament in the former case than in the latter. Rarely a swelling in the right hypochondrium is caused by cancer of the duodenum or of the hepatic flexure of the colon; one in the left hypochondrium by cancer of the splenic flexure. But the commonest site for cancer of the bowel is the rectum or sigmoid flexure. It is important to remember that in malignant disease of the sigmoid or hepatic or splenic flexure the symptoms often point to the cæcum, and there is a great liability to its distension and ulceration, in consequence of which a fatal issue often speedily ensues.

4. A sausage-shaped tumour in the course of the colon, becoming tense and prominent when manipulated or during attacks of griping pain, is characteristic of **intussusception**. The commonest variety of intussusception is the **ileo-cæcal**, in which the ileum and cæcum pass into the colon, the ileo-cæcal valve being foremost. A tumour is first formed in the right flank, it then shifts its position and may be observed to travel to the upper part of the abdomen, and thence downwards to the left flank, where it is usually most conspicuous; sometimes there is a corresponding depression on the opposite side of the abdomen.

A tumour also occurs in about half the cases of **ileo-colic** intussusception (the variety in which the end of the ileum is prolapsed through the ileo-cæcal valve); but a tumour is less frequently associated with intussusceptions affecting the small intestine—the **enteric** variety, or the large intestine—the **colic** and **rectal** varieties.

Splashing and **Gurgling** may be felt and heard in the right iliac region in enteric fever or even in simple intestinal catarrh, and in the left iliac region in cases of dysentery. Abscesses in the neighbourhood of the cæcum occasionally give rise to a feeling of emphysema, owing to the presence of gas in the purulent contents.

The Anus and Rectum.—The anus is to be examined for piles, fissures, fistulæ, condylomata. The rectum is to be carefully explored, with the finger only and bimanually—that is, with the finger of one

hand in the rectum and the other hand placed over the lower part of the abdomen,—whenever there is reason to suspect disease of this part or in its vicinity, and indeed in all cases of obscure disease in any part of the abdomen, for by one or other of these methods the condition of the pelvic organs, the lower lumbar vertebræ, the glands, and any masses in the abdominal cavity below the level of the umbilicus, can be investigated, and in the youngest child as well as in the adult.

First as regards the rectum itself, a narrowing of its calibre may be detected in consequence of (1) congenital, syphilitic, or cancerous disease of its walls, or (2) of pressure as by a tumour from without.

A stricture near the anus is frequently of syphilitic origin; one higher up, associated with irregular nodules or a solid tumour, is usually due to cancer. Care must be taken not to mistake hypertrophy of the prostate, the cervix uteri, hard fæcal masses, or a polypus for malignant disease. Hard fæcal masses may be cleared away by enemata; a polypus is pendant, and readily bleeds when manipulated.

A smooth ring-like mass with a dimple in its centre, feeling somewhat like the os uteri, and becoming tense and firm when the patient strains, is significant of an intussusception, which is commonly of the ileo-cæcal variety; in such a case blood-streaked mucus oozes from the rectum, and the general symptoms are severe.

By bimanual examination the following conditions, amongst others, may be felt in different cases:—(1) Inflammatory thickening about the vermiform appendix, or in connection with the lower portions of the small intestine; (2) tubercular masses or bands in the iliac fossæ, or stretching across the abdomen; (3) cancerous masses; (4) enlargement of the lymphatic glands from tubercular or cancerous deposit.

Thickening of the vesiculæ seminales can be felt through the rectum, and when present suggests the tubercular nature of an otherwise obscure case.

When there is thought to be disease higher up the bowel than the finger can reach, it may be advisable to administer chloroform, and to introduce the whole hand into the rectum.

EXAMINATION OF THE FÆCES.

In all cases of illness the medical attendant should make inquiries with regard to the action of the bowels and the characters of the stools, and whenever possible he should examine the stools himself. The well-being of a patient, from whatever disease he may be suffering, so largely depends upon the state of his alimentary canal that it is essential to obtain full information with regard to the condition of the evacuation.

The following are some of the chief points to be observed in making an examination of the fæces.

Quantity.—This must be distinguished from increased frequency of defæcation, which may occur with the passage of but little fæcal matter. The amount of the stools varies with the quantity and kind of food taken; thus it is larger on a vegetable than on an animal diet. It is also increased in diarrhœa, and especially in cholera. Large quantities of firm, dry motions may be passed after prolonged constipation.

Colour.—The colour of healthy fæces varies to some extent according to the kind of food eaten. Thus it is light yellow on a milk diet, and dark brown when much meat has been taken. It is also much modified by the administration of certain drugs. For example, rhubarb, senna, and santonin tend to make the motions bright yellow, iron and bismuth blacken them, while calomel gives them a green, logwood a reddish-brown, and iodine a bluish tint. If the influence of food and drugs can be excluded, then any change in colour is probably dependent on variations in the quantity or quality of bile present in the fæces, or on the entrance of blood or pus into the intestinal canal.

Greenish stools are common in the acute intestinal catarrhs of infancy, and in the early stages of many cases of profuse diarrhœa. The colour indicates that bile is present in excess or has undergone changes either within the intestine or after the expulsion of the fæces; for example, the yellow stool of a child may turn green on exposure to the air. Sometimes a green stool contains small masses of a yellowish white colour; these are composed principally of fat, as proved by their solubility in ether. Such masses are to be distinguished from the lumps of casein which are so common in the stools of infants. The latter are hard, white, cheesy-looking lumps, and indicate that milk has been imperfectly digested.

Fatty stools are often found in association with disease of the pancreas, but are by no means characteristic of it.

White, clayey, or drab-coloured stools occur in cases of obstructive jaundice or of deficient bile formation. White stools in the absence of jaundice may be due to disease of the pancreas, but occur also in other morbid conditions. The stools tend to become pale when there has been profuse diarrhœa for some time, as in cases of Asiatic cholera.

Blood may give a red, reddish-brown, or black colour to the stools. When it comes from the rectum blood usually has its natural red colour, and streaks or covers the surface of the stool, which otherwise may be unaltered in character. When it enters the bowel at a higher level it undergoes changes and becomes more or less intimately mixed with the fæces, so that a dark brown or blackish pulpy mass is passed. To the passage of such stools the name **melæna** is given. The chief

causes of melæna are ulcerations or intense venous congestion of the stomach or bowels.

Reaction.—The reaction of normal fæces appears to be variable. The albuminous disintegration which occurs in typhoid and in certain forms of dyspepsia leads to decided alkalinity, whereas in the acute catarrhal enteritis of early life the reaction is usually acid. Offensive putty-like stools are commonly alkaline.

Odour.—The stools of infants at the breast have a sour odour. This happens too in some cases of infantile catarrh, while in other cases the motions have a highly offensive, cadaverous fœtor. The latter is also often noticeable when there is dysenteric, syphilitic, or cancerous disease of the rectum. Typhoid stools are often very offensive.

Form and Consistence.—Liquid stools are found in the various forms of diarrhœa, and are particularly copious and frequent in Asiatic cholera. In the choleraic diarrhœa of infancy the stools are loose and frothy at first, and of a yellowish or greenish colour; in the later stages they become quite watery, and may resemble the rice-water evacuations of cholera. In the severest cases they consist of a brown-coloured offensive watery liquid. The "rice-water" stools of *cholera* look like water in which rice has been boiled, they are quite thin, and may be entirely devoid of smell or colour. Stools of similar character occur also in cases of acute arsenical poisoning, and for the diagnosis of Asiatic cholera it is necessary to separate and make cultivation of the comma bacillus. The stools of *typhoid fever* present a resemblance in colour and consistence to pea-soup. They contain particles of undigested food, bile-stained mucus and epithelial cells, crystals of triple phosphates, and numerous fungi; while flocculent shreds and fragments of sloughs may be discovered during the third or fourth week of the disease. Sometimes loose liquid stools contain globular, hard fæcal masses of various sizes. These **scybala** suggest a condition of fæcal accumulation, the diarrhœa being excited by the irritation of hardened fæces. When the lower part of the bowel is narrowed as by a cancerous stricture, narrow, flat, or ribbon-shaped motions may be passed; also, fæcal masses may be grooved by the presence of a rectal polypus.

Constituents.—In order to examine the various constituents of a stool, the following method should be adopted. If the stool is quite liquid, a portion of it should be poured into a large conical glass vessel. If it is solid, it should be washed with a stream of water until it is completely broken up, the whole being then poured on to a fine sieve placed over the conical glass vessel. By this simple method any abnormal constituents, such as particles of undigested food, foreign bodies

accidentally swallowed, shreds of membrane, worms, or gall stones can be satisfactorily inspected; while by means of a pipette a few drops of the liquid material may be drawn up from the bottom of the glass, mounted on a glass slide, and examined under the microscope.

The appearance of blood in the stools has been already referred to, and a few observations may now be made with regard to the presence of mucus, pus and worms; but for a detailed account of the microscopical and chemical examination of the fæces, we must refer the student to special works.

Mucus.—When particles, shreds, or masses of mucus are distinctly recognised, the intestinal mucous membrane is usually in a state of catarrh. Large shreds, membranes, or jelly-like masses of mucus indicate catarrh of the rectum or lower part of the colon. They are frequently streaked with blood. In some cases they are passed without admixture of fæces, in other cases the latter are enveloped by a thin

FIG. 175.—Tænia saginata: head; proglottis; egg; magnified. (*v. Jaksch.*)

layer of mucoid material. Sometimes shreds of mucus are called "skins" by the patient, and he may mistake them for worms.

If mucus comes from the small intestine or upper portion of the colon, it is usually intimately mingled with the fæces, and cannot be detected without the aid of the microscope. If particles of mucus are found to be stained yellow with bile pigment, it is probable that the small intestine is involved.

Pus in the motions may come from a fistula, cancer, or other lesion of the rectum, or from a pelvic, prostatic, or other abscess which has opened into the bowel. Muco-purulent stools are also found in cases of chronic catarrh affecting the lower bowel.

Of the various worms that inhabit the intestinal canal, the following are probably the commonest:—

Tapeworms.—The **Tænia mediocanellata,** or saginata, is the most frequently met with in this country. The head has a central groove

or depression, and is furnished with four large suckers, which are generally much pigmented. It has neither rostellum nor hooklets. The ripe segments or proglottides which are passed in the stools are oblong in shape, and of an opaque white colour. The uterus is very much branched, and the genital pore projects from one side of the proglottis.

The **Tænia solium** is usually shorter than the tænia mediocanellata.

FIG. 176.—Tænia solium: head, magnified; proglottis, actual size; egg magnified. (*v. Jaksch.*)

Its head is dark in colour, and about the size of a pin's head; it has four lateral suckers, and between them a rounded projection or rostellum, which is surrounded by a double row of hooklets, about twenty-six in number. The uterus is less branched than that of the preceding worm; its genital pore is situated at the side of the proglottis.

The **Bothriocephalus latus.**—In this worm the sexual openings occur

FIG. 177.—Head of Bothriocephalus latus, magnified: *a*, seen on edge; *b*, seen on the flat; *c*, proglottides; *d*, eggs.

on the surfaces of the segments, and not at the sides, as in the case of the preceding worms. The head is ovoid, cleft, and provided with two lateral suckers, but has no hooklets or rostellum. In seeking for the head or for the eggs of a tapeworm, the stools should be treated in the way already mentioned. The head and the long thin neck may be seen with the naked eye, but for the detection of the ova a microscope is necessary.

Roundworms.—(*a.*) The **Ascaris lumbricoides** has a shape similar to that of the common earth-worm. The head is distinct from the body, and has three lateral papillæ. The body is cylindrical, and tapers at both ends, especially towards the tail end. The female is much longer than the male. This worm infests the small intestine, and sometimes finds its way into the stomach and is expelled by vomiting. Occasionally it makes its way into the common bile duct.

(*b.*) The **Oxyuris vermicularis**, or thread-worm, inhabits the rectum and descending colon. The male is about one-sixth of an inch in length, the female about half an inch. These worms often produce much itching at the anus, and also about the vagina or prepuce. There may be some vaginal discharge or irritability of the bladder, and frequent micturition, or tenesmus and prolapsus ani.

(*c.*) **Anchylostomum duodenale.**—The presence of this parasite should be suspected in cases of severe anæmia occurring in epidemic form when there is no obvious or sufficient cause. The female measures a little more than half an inch in length; the male about one-third of an inch. Unless anthelmintics have been given, the eggs alone may be found in the evacuations. The eggs are oval, have a clear shell and granular contents, often showing cell division.

(*d.*) The **Tricocephalus dispar**, or **Whipworm**, is stated to occur in great numbers in beri-beri. It is distinguished by its short stout hinder part, and by the spiral filiform character of its anterior extremity. It inhabits the cæcum, and is usually from one and a half to two inches in length.

(*e.*) The **Trichina spiralis**, about $\frac{1}{25}$ of an inch long, develops in the intestinal canal, but ultimately reaches the voluntary muscles of the body, where it is found in enormous numbers. The chief symptoms, viz., pyrexia, limb pains and tender swollen muscles, may be ushered in by signs of gastro-intestinal disturbance.

THE LIVER AND GALL BLADDER.

Of the numerous and various symptoms that may be produced by diseases of the liver and gall bladder, the most important are pain, jaundice and ascites. Some points bearing on these symptoms have been already noticed, but before proceeding to consider the physical signs that may be observed during an examination of the hepatic region, it is desirable to make a few observations with regard to hepatic pain and jaundice.

Hepatic Pain.—Perhaps the commonest variety of pain produced

by disorders of the liver, is a heavy feeling of discomfort or distension in the right hypochondrium. This is often accompanied by a referred pain in the right shoulder, which is usually most marked in the neighbourhood of the inferior angle of the scapula, and which sometimes extends across the spine to the left scapula. When the surface of the liver is diseased, as by an abscess or a perihepatitis, the shoulder-tip pain of phrenic irritation may also be present. In perihepatitis, pain over the liver is frequently constant and severe. But the severest pain of all is that known as biliary colic. This is most commonly caused by the passage of a gall stone along the cystic or the common bile duct, but may occur when the opening of the bile duct into the duodenum is obstructed by an ulcer or other lesion. Pain often abates in intensity when a stone passes from the cystic into the larger common duct, but again becomes severe when it reaches the duodenal orifice, while its escape into the duodenum is accompanied by instant relief. An aneurysm of the hepatic artery is a rare cause of intermittent paroxysms of neuralgic pain, which sometimes closely simulate attacks of gall-stone colic.

Jaundice.—The causes of jaundice may be tabulated as follows:—

1. **Obstruction of the Hepatic or of the Common Bile Duct.**—Gall stones or inspissated bile; hydatids, distomata; foreign bodies from the intestines; inflammatory swelling of the duodenum, or of the wall of the bile duct; stricture or obliteration of the duct from congenital defect, perihepatitis, former ulceration of the duodenum or of the bile ducts; compression of the duct by tumours of the liver, pancreas, stomach, kidney, omentum, ovary or uterus, by an abdominal aneurysm, or by enlarged glands in the portal fissure.

2. **Cirrhosis and the Various Forms of Chronic Atrophy of the Liver.**—Jaundice in these cases is probably caused by obstruction of the small bile ducts in the liver.

3. **Poisons.**—Phosphorus, mercury, arsenic, antimony, copper, chloroform; snake poison.

4. **Vascular Changes in the Liver,** as in the chronic congestion from heart disease.

5. **Disturbance of the Nervous System.**—Concussion of the brain; mental emotions.

6. (1) **The Specific Fevers**—yellow fever, malarial fevers, relapsing fever, typhus, enteric fever, scarlatina and pyæmia; (2) **acute pneumonia**; (3) **acute yellow atrophy.**

In group 1, when the larger ducts are completely obstructed the motions are white and clayey; but in the other groups the stools may be normally bile-stained.

The commonest cases of jaundice are those known as **simple** or

catarrhal. The patient is usually young, and has suffered from symptoms of dyspepsia. Frequently there is no abdominal tenderness, and nothing to be made out by palpation over the edge of the liver, but occasionally tenderness and increased resistance to palpation at the right costal margin do exist. When jaundice is **associated with ascites**, the disease is probably cancer or cirrhosis of the liver; jaundice is usually more intense in cancer than in cirrhosis. When jaundice is **immediately preceded by attacks of severe paroxysmal pain** in the hepatic region, it is probable that a gall stone has passed along the cystic duct into the common duct. The cessation of the pain and the gradual subsidence of the jaundice would indicate that the stone had passed into the duodenum. The coexistence of jaundice and paroxysmal pain occurs also in cases of cancer, and rarely in hydatids of the liver, or in aneurysm of the hepatic artery. The **sudden onset** of jaundice in the midst of good health suggests obstruction of the common duct by a gall stone or other foreign body, while the **slow onset** and gradual development of intense jaundice suggests either the growth of some tumour in the duct or pressure on it from without. The coexistence of **pyrexia** indicates an acute febrile disease, as one of the specific fevers or pyæmia, or suppurations within the liver. Temporary fever, however, may accompany the passage of a gall stone.

The above statements illustrate the importance of studying the associations of jaundice, its mode of onset, and the order in which the various symptoms are developed. The age and previous health of the patient are also factors which aid in the diagnosis of the cause of any case of jaundice.

Anatomical Relations.—Three-fourths of the liver lie to the right, one-fourth to the left, of the middle line of the body. The highest point of its convex upper surface reaches the level of a plane drawn through the sternal ends of the fifth costal cartilages, and the body of the ninth dorsal vertebra, and is situated opposite the fourth right costal space, midway between the mammary and parasternal lines. This is about three finger's breadths higher than the lower edge of the right lung. The left lobe of the liver lies in front of the stomach and behind that portion of the diaphragm on which the heart rests. The lower sharp edge of the liver passes down and out from the twelfth dorsal vertebra along the eleventh rib towards the axilla. In the mammary line it lies just beneath the costal arch; while in the epigastrium, an average position for the lower margin is about half way between the xiphoid cartilage and the umbilicus.

The lower margin of the left lobe begins in the middle line, and passes obliquely upwards to reach the left costal margin at the junction of the seventh and eighth cartilages; behind the cartilages it is con-

tinued as far as the heart, and terminates a little to the right of its apex beat.

The notch of the gall-bladder is situated just at the point where the lower edge of the liver leaves the right costal arch.

The liver in young children is larger proportionately in all its dimensions than in the adult, and its lower edge may be found below the costal margin in the axillary line.

Inspection.—In the healthy adult no difference is to be seen between the right hypochondrium and the left, but sometimes in infants the former is more prominent than the latter.

A marked enlargement of the liver, or its downward dislocation often produces a visible prominence of the right hypochondriac or epigastric region. In infants, bulging of the costal cartilages readily occurs; and in their abdomens it is sometimes possible to see a projection caused by the lower edge of an enlarged liver, when its sinking and rising during inspiration and expiration may be observed.

Palpation.—This is by far the most valuable method of examination in the investigation of liver diseases. The palmar surface of the fingers should be laid gently and evenly on the abdomen some distance below the right costal margin, the finger-tips being inclined upwards and slightly inwards towards the median line; they should then be pressed in with moderate firmness while the patient takes a deep breath; if now the edge of the liver be lower than normal, it will be felt to descend beneath and float up the fingers.

But when the abdomen is tense, as from much ascites or meteorism, the process of "dipping" is often the only method of reaching the liver; this consists in applying the fingers perpendicularly to the surface and then pressing in the tips suddenly and forcibly, when the solid organ may be felt, and often at the same time a sense of displacement of fluid.

In health, the edge of the liver may sometimes be felt in children, and in women whose abdomens are relaxed by child-bearing; but in men, as a rule, there is merely some increase in resistance to be detected in the epigastrium during a deep inspiration.

In nearly every case, then, if the edge of the liver is distinctly felt below the costal arch, the organ is either enlarged or displaced downwards. A distinction between enlargement and displacement is usually easily made, but sometimes a diagnosis is difficult, and requires a knowledge of the causes which lead to these conditions, as well as a consideration of the associated condition of other organs. It is also necessary to mention that enlargement and displacement may coexist in the same case.

Displacement Downwards may result from thoracic deformity, and

is especially noticeable in severe rickets. The liver is also pushed down by tight lacing, by right pleural effusion, right pneumo-thorax, and to a less degree by effusion into the left pleura, by emphysema, by pericardial effusion, by enlargement of the heart, and sometimes by an abscess, hydatid, or other swelling, situated between the liver and diaphragm.

In rare cases, owing to relaxation of the suspensory ligaments and to overstrain, as in severe labour, the liver may hang down very low, even as far as the iliac region. It is then called, on account of the ease with which its position may be altered, a *movable* or *wandering* liver.

Enlargements of the liver are conveniently divided into two classes, according to the presence or absence of local tenderness.

1. **Painful on Palpation.**—Painful enlargements result from one of the following causes :—

Congestion, especially if quickly produced.
Interstitial hepatitis.
Hypertrophic cirrhosis.
Retention of bile from catarrh of the bile ducts, or from obstruction of the common duct.
Abscess.
New growths, such as cancer, lymphosarcoma and gummata. The local tenderness varies according to the degree to which the peritoneum is implicated.

2. **Painless on Palpation.**—The painless enlargements comprise :—

The fatty liver.
The amyloid liver.
Hydatid disease of the liver.

Jaundice and ascites are frequently found in association with group 1, but not, as a rule, with group 2.

Size.—Cancer, amyloid degeneration and hydatid disease give rise to the greatest enlargements. The enlargement is uniform in all acute and chronic enlargements, with the exception of those due to abscess, new growths, syphilis and hydatid disease, in which certain parts of the liver are alone affected.

Shape and Character of Surface.—A tense, round, smooth swelling, usually implicating the right lobe of the liver, indicates an abscess, often of tropical origin, or a hydatid cyst; the former is tender on pressure, the latter not. In cancer, large nodules and ridges, or small rounded elevations, with cup-shaped central depressions, are often to be felt (see Fig. 178). The projections caused by syphilitic gummata are usually smoother and flatter than those of cancer, and they may be associated with scar-like depressions on the surface of the liver. A rough granular surface is characteristic of cirrhosis, but the unevenness

of the surface may be out of reach of the finger, owing either to ascites or to the diminished size of the liver.

Consistence.—The fatty liver is soft and flabby, and its edge is rounded. The congested, cirrhotic and amyloid livers are firmer than natural; the edge of the amyloid is rounded, but that of interstitial hepatitis and of hypertrophic cirrhosis is sharper as well as harder than natural.

Fluctuation, or an elastic sensation, is felt over a hydatid cyst or an

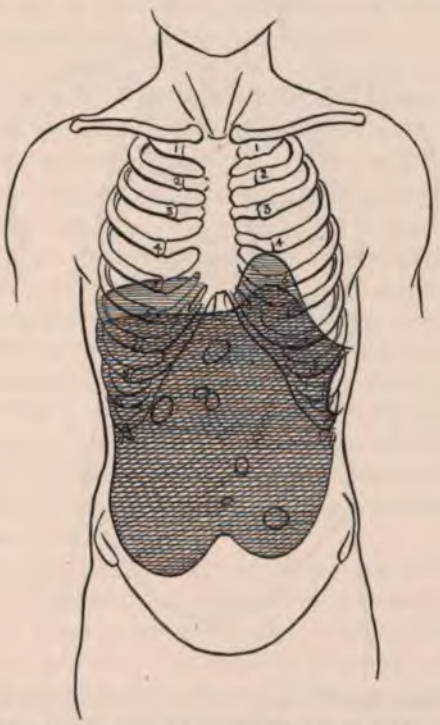


FIG. 178.—Cancer of the Liver—extreme enlargement; the small circular outlines indicate the position of nodules which felt hollow on the surface.

abscess, according to their depth from the surface. The "hydatid thrill," a peculiarly long tremulous sensation, is conveyed to the left hand, laid flat on the echinococcus tumour, on percussing the latter with the fingers of the right hand. This thrill, however, is also met with over ovarian or other cysts, and, rarely, even in cases of ascites.

Influence of Respiratory Movements.—All enlargements of the liver, and all tumours connected with it, rise and fall with inspiration and expiration. Sometimes such movements are limited by adhesions (as in perihepatitis), by tumours in other abdominal organs, by meteorism

or ascites, or owing to very great enlargement of the liver. Occasionally during respiration *friction fremitus* may be felt over the surface of the liver; this may also be sometimes produced by pushing the abdominal wall over the liver.

The **Gall-Bladder**, under normal conditions, is very rarely to be felt. When enlarged, it is recognised by its pyriform or globular shape, its elasticity, and its relation to the edge of the liver. Obstruc-

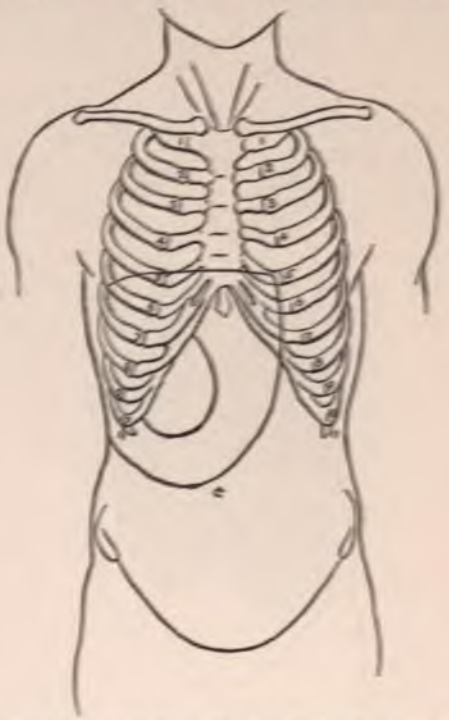


FIG. 173.—Case of Hydatid Tumour of Liver. The outer outline represents the size of the liver as determined by palpation and percussion; the inner outline the position of a smooth globular swelling, which projected from anterior surface of liver. On puncturing the tumour, a pale yellow liquid obtained, sp. gr. 1.005; a trace of albumin; booklets seen under the microscope. (*Dr. Sturgess's Case.*)

tion of the bile-duct is followed by distension of the gall-bladder, which projects as a large tense tumour below the edge of the liver, nearly in the mammary line. This may become inflamed and suppurate, and is then the seat of much pain and local tenderness; in such a case the patient is feverish, and often suffers from rigors and night sweats.

A hard and sometimes nodulated **tumour** at the site of the gall bladder indicates a collection of stones in the gall-bladder, or cancerous disease of its walls. In the former case the tumour is usually movable, in the latter immovable. The two conditions are often associated,

and not infrequently gall-stones precede the development of cancer of the liver or gall-bladder.

A sensation of **crackling** on pressing over an enlarged gall-bladder may be due to the presence of calculi, or of softening in the interior of hard nodules of cancer.

This is a convenient place to consider some of the **Pathological Consequences of Gall-Stones.**

1. There may be a large collection of stones in the gall-bladder without any history of jaundice, sometimes indeed of any symptoms. Nor is the gall-bladder necessarily much enlarged.

2. When a stone is impacted in the cystic duct, jaundice may be absent, but symptoms of biliary colic are usually present.

3. A stone may form in the biliary ducts within the liver, and then become lodged in the hepatic duct, when there will be symptoms of obstructive jaundice without enlargement of the gall bladder.

4. An impacted stone in the common duct may be a cause of permanent jaundice. The symptoms bear some relation to the shape of the stone; thus an angular stone may cause severe pain, but may allow bile to trickle past it, and hence jaundice may be slight or absent.

5. Gall-stones which have entered the intestine may be voided per anum, or may become impacted in the bowel, and cause symptoms of intestinal obstruction. In the latter case the calculus usually gains entrance to the bowel by means of a fistulous communication with the gall-bladder.

6. Gall-stones may set up inflammation and ulceration of the mucous membrane of the gall-bladder or bile ducts, and so lead to pyæmia or to perforation and peritonitis.

In other cases ulceration or sloughing of the wall of the gall-bladder is attended by the formation of adhesions to the stomach, duodenum, or colon, when a fistulous communication forms between them, and the stone or stones pass into the stomach or bowel. Sometimes gall-stones may be discharged from the gall-bladder or bile-ducts through fistulous openings in the abdominal parietes.

Percussion.—The **superficial** or **absolute dulness** corresponds to that part of the liver which lies directly in contact with the thoracic and abdominal walls. A stroke of extreme lightness is requisite to map out this area, which has the following limits:—

The Upper Boundary.		*The Lower Boundary.*
Base of xiphoid cartilage	in middle line	nearly half way between xiphoid cartilage and umbilicus.
The 6th rib	in mammary line	costal margin.
The 8th rib	in axillary line	10th rib.
The 10th rib	in scapular line.	11th rib.
The 11th rib	near the spine.	

The line marking the **upper boundary** is slightly convex downwards; that part of it which lies to the left of the sternum is difficult to distinguish from the cardiac dulness, and, as a rule, it is sufficient when mapping out the upper limits of the heart and liver to draw a continuous line, connecting across the sternum the cardiac with the hepatic dulness (see Fig. 180).

The **lower boundary** of dulness, which corresponds to the lower edge of the liver, cannot be made out behind between the spine and the scapular line, because there the dull note of the liver is continuous with that over the kidney and lumbar muscles. In the middle line in front also it is difficult to separate the liver from the colon; the former gives a muffled, the latter a clear tympanitic note, and a very gentle percussion stroke is necessary to distinguish the one from the other. It is obvious, too, that the degree of accuracy with which the edge can be made out by percussion will vary greatly with the tension of the abdominal wall, and with the degree of distension of the stomach or intestines. On percussing out the extreme left portion of the lower boundary, it will be found to pass in some cases directly into the superficial cardiac dulness, in other cases into the edge of the lung —the former is of frequent occurrence in children, the latter in adults. One indication of enlargement of the left lobe is that it abuts against a larger corner of pulmonary resonance than ever occurs under normal conditions.

The superficial hepatic dulness is diminished during a deep inspiration, and increased during a deep expiration. This is due to the fact that the lower edge of the lung moves more than the lower edge of the liver during respiration.

The deep or relative dulness runs parallel to and about one inch and a quarter higher than the upper line of superficial dulness. On percussing downwards from below the clavicle in the parasternal line, the comparative dulness is first detected at the fifth cartilage, or in the upper portion of the fifth interspace. This is a little lower than the top of the liver, which is covered by too great a thickness of lung to modify the sound elicited by even forcible percussion. Still, if we very carefully observe the slightest modification in the pulmonary resonance, we may often map out pretty accurately the upper limit of the liver, as proved by postmortem examination. Behind, the relative dulness is more difficult to make out, and can rarely be of much service in diagnosis.

Liver Dulness in Diseased Conditions.

Area of dulness diminished.—This is due to one of the following causes:—

1. Tympanitic distension of the stomach or colon may separate the liver from the abdominal wall or tilt up its lower edge.

2. Pressure from below is the most frequent cause, as in cases of large ovarian or omental tumours, or when there is great ascites.

3. A diminution in the size of the liver, as from acute yellow atrophy, or alcoholic or syphilitic cirrhosis.

But it is to be noted that a diminution in hepatic dulness indicates a small liver only when the upper limit of superficial dulness is not higher than normal, and when all those conditions which push up the liver and diaphragm can be excluded. In acute yellow atrophy the area of dulness is reduced with great rapidity, not only because the

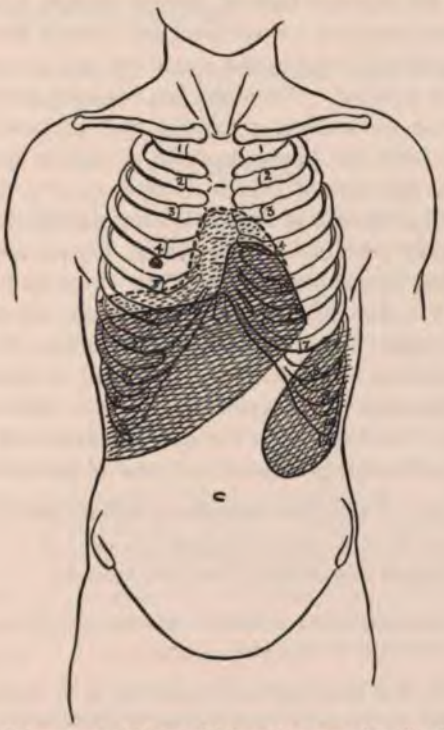


FIG. 180.—Enlargement of Liver and Spleen, in a case of Cardiac Dilatation.

liver becomes smaller, but because owing to its softness it falls backwards, and allows the intestines to ride over it. When there is much atrophy the dulness disappears from the epigastrium; and it may be observed that evidence of contraction of the liver is often first manifest to the left of the middle line, for the left lobe is usually, and especially in syphilitic cirrhosis, more affected than the right lobe. Absence of the "absolute" dulness in the mammary or in the axillary line, is usually due to distension of the colon with gas, or to an altered position of the liver.

In cases of moderate emphysema where the area of the diaphragm is not increased, the absolute liver dulness may be diminished while the relative is increased. But in great emphysema the relative dulness becomes less or disappears altogether while the absolute, though lower in position, maintains its normal depth.

Area of dulness enlarged. — While a diminution in the area of hepatic dulness is due often to interposition of the lung, it may also be caused by other conditions which will require to be considered. An increased dulness which is caused in the majority of cases by enlargement of the liver. Unless the enlargement is great the anterior limit of dulness remains unaltered, whereas the inferior limit extends in a lower and sometimes further to the left, where it may obtain the edge of the mammary line, or even pass directly into the splenic dulness. When the liver is very greatly enlarged, or when a large tumour, such as a hydatid cyst projects from its upper surface, it is possible for the dulness to reach as far as the second rib.

An Apparent Increase of hepatic dulness occurs —**Upwards** in consequence of : 1. an effusion of liquid into the right pleura, or consolidation of the lower part of the right lung ; 2. an abscess or tumour situated between the upper surface of the liver and the diaphragm. **Downwards** : 1. when the stomach and colon are filled with solid material ; 2. when there is strong contraction of the abdominal muscles ; 3. in cases of retraction of the chest in great emphysema, or in the neighbourhood of the pleurisy ; 4. when there are projections from the edge of the liver as in the case of a distended gall-bladder.

The results of hepatic percussion may also be grouped as follows :—

The lower edge of the liver occupies a normal position.

A. The upper limit of dulness is **higher** than normal.

1. There is an enlargement of the liver upwards.

or—

2. There is excessive pneumo-thorax effusion, the dulness of which passes into that of the liver.

or

3. The liver is enlarged and pushed up by an ascites.

B. The upper limit is **lower** than normal, when there is a moderate degree of emphysema.

The lower edge is lower than normal.

A. The upper limit of dulness is normal.

The liver is enlarged.

B. The upper limit is higher than normal.

(1.) There is great enlargement of the liver.

(2.) There is much pneumo-thorax effusion.

(3.) There is a large swelling between the liver and the diaphragm.

C. The upper limit is lower than normal.

(1.) There is much emphysema.

(2.) There is pneumo-thorax.

The **lower edge is higher than normal.**

A. The upper limit is **higher** than normal.
The liver is dislocated upwards.

B. The upper limit is **normal.**
(1.) The liver is smaller than natural.
(2.) There is ascites, or meteorism, or air, in the peritoneal cavity.

No liver dulness can be detected.

A. There is much ascites, or there is gas in the intestines or in the peritoneal cavity.

or—

B. There is extreme atrophy of liver.

or—

C. The liver occupies an abnormal position owing either to its mobility or to congenital displacement.

THE SPLEEN.

Anatomical Relations.—The spleen, somewhat oval in shape, lies deeply in the left hypochondrium. Its long diameter is directed downwards and forwards, the upper edge corresponding pretty closely with the ninth rib, and the lower edge with the eleventh rib; the lower margin overlaps the top of the left kidney. The upper and posterior part of the spleen is covered by lung, and its hinder end approaches the body of the tenth dorsal vertebra. The anterior end reaches nearly as far as the mid-axillary line; it is opposite to the free end of the eleventh rib, and does not cross a line drawn between this and the left sterno-clavicular articulation "costo-articular line." The upper edge, which commonly presents one or two notches, constitutes the outer boundary of Traube's semi-lunar space, and forms a sharp angle with the lower border of the left lung (see Fig. 183.) The anterior end of the lower edge is about one inch above the costal margin. Only the lower two-thirds of the spleen are in direct contact with the chest wall. The organ is proportionably larger, and is situated a little further back in the child than in the adult. After the age of forty it begins to diminish in size.

Inspection.—Ordinarily no information is obtained by inspection of the splenic region. It is only when the organ is very greatly enlarged that the left hypochondrium and the abdominal wall situated below and in front of it appear unduly prominent. Then possibly the fulness may be observed to rise and fall during the respiratory movements.

Palpation.—The spleen can only be felt when it is pushed down below the ribs, or when it is enlarged.

Downward displacement occurs in infants with rickety chests;

sometimes from large effusions into the left pleura; and rarely from emphysema. Exceptionally its ligaments become relaxed, and permit the organ to escape from its normal position and to become a "**movable spleen**;" it may then be found in very unusual situations—for example, in a hernial tumour in the inguinal region.

The Spleen is Enlarged—

1. In certain of the acute specific fevers, especially in enteric fever, in relapsing fever, and in ague. A slight enlargement is sometimes manifest in erysipelas and in pneumonia.

FIG. 161.—Great Enlargement of Spleen. Photograph of a man, the subject of leucocythæmia, who formerly had suffered from many attacks of ague. (Dr. Simpson's Case.)

2. In tuberculosis, in leucocythæmia (which gives rise to the greatest enlargement), in some anæmic conditions, in lymphadenoma (Hodgkin's disease), and in amyloid degeneration.

3. In association with some cases of rickets, but not directly depending on this disease. From syphilis either hereditary or acquired.

4. From embolism in ulcerative endocarditis, and sometimes in cardiac disease (congested spleen), apart from embolism; by the presence of abscesses, whether pyæmic in origin or the result of ordinary inflammation. Large solitary abscesses are commoner in the liver than in the spleen, where they are very rare.

5. By a new growth—gumma, cancer, lympho-sarcoma.
6. By hydatid or other cysts.

As a rule, an enlarged spleen is easily felt; it projects as an oval solid tumour from below the left costal arch, and passes downwards towards the iliac crest, and forwards towards the middle line. It is superficial and mobile, and can be felt to descend with each inspiration. The upper or anterior margin is usually thin, and presents a well-defined notch; sometimes there are two, and occasionally even four notches. The lower or posterior edge feels rounded, and is quite free, the fingers being easily introduced between the spleen and the erector spinæ.

Method of Palpating.—When the spleen is only moderately enlarged, or when, owing to its softness, a doubtful result is obtained by palpating the patient in the ordinary position, the following method is helpful. The patient should lie half way between a dorsal and a right-side position, so that he rests on the right shoulder-blade and the right hip; the left knee should be drawn up, and the patient should take a deep inspiration from time to time. The examiner, standing either on the patient's right or behind him on his left side, palpates with the first three fingers of his right hand. In the former position he should pass them upwards and a little backwards towards the intervals between the cartilage of the tenth rib and the free end of the eleventh rib. In the latter position the fingers are to be pressed in vertically to the plane of the body. The bimanual method, in which one hand is placed behind and the other in front, is also frequently of value.

Fallacies.—(1.) An enlarged spleen, if very soft, may only convey a sense of increased fulness or resistance to the examining hand. This is the case sometimes in typhoid fever.

(2.) In the absence of enlargement or displacement of the spleen, it sometimes happens that when pressing deeply into a flaccid abdomen the contraction and descent of the diaphragm gives a similar feeling to that of the spleen. The last rib, too, may easily be mistaken for the edge of the spleen.

Tender on Palpation.—When the enlargement is due to rapid congestion, to infarcts, or to abscesses, there is often much tenderness. A cancerous spleen, a rare condition, is usually painful on pressure.

Size.—The largest spleen occurs in leucocythæmia, in which disease it may extend into the right side of the abdomen. In typhoid fever the organ does not project much below the ribs. In hereditary syphilis the enlargement may be observed at the age of two or three months; it may persist and increase in size, so as to be considerable when other signs of syphilis have quite vanished.

Surface.—The surface of an enlarged spleen is usually smooth and

even. In leukæmia flat projections, in cancer hard nodular tumours, and in cysts or abscesses rounded elastic swellings may be perceptible. *Friction fremitus* may be felt when the peritoneal coat is inflamed. *Pulsation* is occasionally present in cases of aortic regurgitation.

Consistence.—This is usually soft in the enlargements that result from the acute specifics, or from passive congestion; but firm and dense in leucocythæmia and in intermittent fever.

Percussion.—A gentle percussion stroke is required to map out

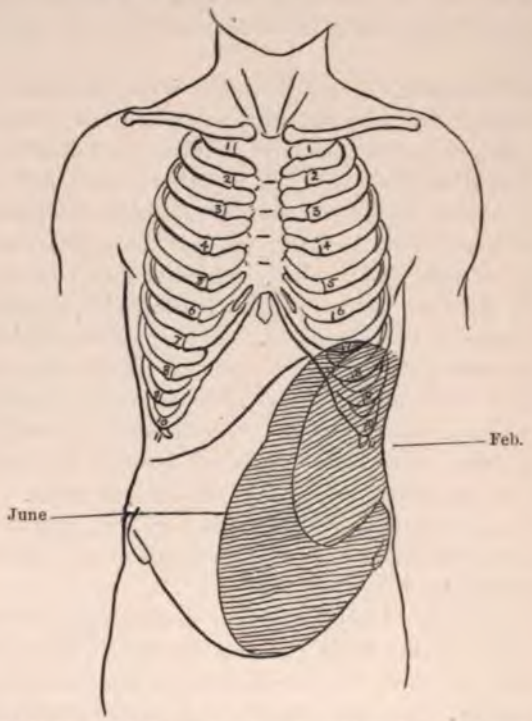


FIG. 182.—Great Enlargement of Spleen in a boy, the subject of marked anæmia; no enlargement of glands. Death from intestinal ulceration—*not* typhoidal; the splenic pulp crowded with large cells containing red blood corpuscles. (Williamson, *Med. Chron.*, 1893.)

the dulness yielded by a spleen of normal size or by one only slightly enlarged. It is better for the patient's body to be erect, or to be slightly inclined towards the right side during this examination. The area of dulness is limited to that portion of the organ which is uncovered by lung. It is situated between the mid and posterior axillary lines extending from the upper border of the ninth to the lower border of the eleventh rib. The outline of the dulness is rounded in front and below, but flattened above where it is bounded by the lower edge

of the left lung. Posteriorly the splenic dulness passes into that obtained over the kidney and lumbar muscles. Diagonally it measures about 3–4, and vertically about 2 inches; anteriorly it is fully 1 inch above the costal margin, and in this direction should not cross the "costo-articular line." The splenic dulness is **diminished or abolished by** a deep inspiration, by emphysema, by distension of the stomach, colon, or peritoneal sac with gas, and in the rare cases where the spleen is either absent or has wandered from its normal position.

The splenic dulness is **apparently increased** by consolidation of the left lung, by a liquid effusion into the cavity of the left pleura, or by the presence of solid material in the stomach or colon. A **real increase** in the area of dulness is due to enlargement of the spleen; then the outline of dulness preserves its normal shape, but is increased in size; and as the spleen becomes also thicker the dulness is more intense, and is accompanied by an increase in resistance.

When the spleen is large enough to project below the ribs, palpation is obviously a more accurate method of examination than percussion; the latter is, however, still of service, for any doubt with regard to the nature of a tumour projecting from the left costal arch is removed if we find that dulness obtained by percussing over it is continued upwards over that part of the chest wall which corresponds to the splenic area.

THE PANCREAS.

Anatomical Relations.—The pancreas, deeply placed in the abdomen, lies in front of the aorta and behind the stomach; it corresponds posteriorly to the level of the first lumbar vertebra, and anteriorly to a transverse line drawn about three inches above the umbilicus. Its head rests in the curve formed by the duodenum, while its tail is in contact with the spleen. Above it is the cœliac axis, and behind its head are the inferior vena cava, the vena portæ and the common bile duct.

The **diagnosis** of diseases of this organ is usually very difficult. The pancreatic region is most satisfactorily examined when the stomach and colon are empty, and the abdominal walls rendered as lax as possible.

Cancer of the head sometimes gives rise to a tumour situated to the right of the middle line and above the level of the umbilicus. The swelling is usually fixed, feels hard and nodular, does not move with respiration, and is placed deeply beneath the edge of the liver.

Jaundice is often present from obstruction of the common bile duct; there may be ascites, and an abundant discharge of fatty matter in the stools is not uncommon. The pancreatic duct may be implicated

without the common bile duct; then, according to Dr. Walker, there are continuously white stools without jaundice.

A **cyst** of the pancreas often reaches a large size, and forms a rounded fluctuating tumour in the upper part of the abdomen. The fluid withdrawn by aspiration is turbid, alkaline, of sp. gr. 1010–1020; contains sugar, albumin, mucin and a trace of urea; and it has the property of emulsifying fat and digesting starch.

THE OMENTUM.

Tumours of the omentum, produced by inflammatory thickening, abscess, tubercular or cancerous deposits, are felt as irregular lobular masses in the lower epigastric and umbilical regions. They are superficial, usually dull to percussion, and, unless adherent to the abdominal wall, descend a little during inspiration. **Tubercular deposits** are not uncommon in the tubercular peritonitis of childhood; they form firm lumps above the umbilicus, or sausage-shaped tumours, which may stretch across from one hypochondrium to the other. **Cancer** of the omentum may simulate cancer of the liver or stomach. From the former it may be distinguished in some cases by a band of resonance between it and the liver, from the latter by a careful examination of the stomach both when empty and when distended by inflation.

THE MESENTERY AND RETROPERITONEAL GLANDS.

Tubercular or **cancerous** deposits may present, on palpation, firm masses, which are more or less fixed, and are situated at or below the level of the umbilicus. A slight enlargement of the glands can only be felt when the abdomen is sufficiently flaccid to allow the hand to be pressed deeply in towards the vertebral column. In some cases a glandular mass transmits **pulsation** from the underlying aorta, when the pulsating tumour has to be distinguished from abdominal aneurysm. A digital examination per rectum may detect cancerous masses of glands pressing down on the upper wall of the rectum, and leading in some cases to partial obstruction of the bowel; ascites may coexist in consequence of cancerous disease of the peritoneum.

THE KIDNEYS.

In studying diseases of any particular organ, the necessity for taking a wide view of pathological processes is constantly forced upon us. Disease of one organ cannot be studied apart from its dependence

and effects on the functions of other tissues. These considerations require to be constantly borne in mind when investigating suspected cases of kidney disease.

Inspection, palpation and percussion of the renal region give us some information; an examination of the urine still more information with regard to the condition of the kidneys; but our knowledge will be inadequate till we have examined the heart and arteries, and have investigated all the symptoms of the case, their order of development, their respective predominance, and their relationship to one another.

The chief symptoms from which a patient with kidney disease suffers may in some cases appear, at first sight, to have no connection with the condition of the kidney. Thus, instead of complaints with regard to alterations in the frequency of micturition, or in the quantity and characters of the urine, we may hear of vomiting, purging, headache, giddiness, sudden loss of sight, excessive wakefulness, or we may be summoned to a patient who is in convulsions, or unconscious, or suffering from paroxysmal dyspnœa. These various clinical phenomena, occurring either separately or combined, indicate the condition called "**uræmia**," a condition usually attributed to faulty kidneys, whereby poisonous products are retained in the body, instead of being eliminated.

In some cases lesions may be found to account for the symptoms, but in other cases this is not so. For example, a patient with granular contracted kidneys suddenly becomes comatose, and dies in a few minutes. At the post-mortem, cerebral hæmorrhage may be discovered, but sometimes no brain lesion can be found. Again, changes in the retina will account in some cases for failure of sight, but the latter occurs in kidney disease apart from any detectable ocular lesion.

Moreover dropsy, which is one of the commonest symptoms present in kidney disease, as well as the various clinical phenomena of uræmia, may have a different pathology in different cases. In acute Bright's disease both uræmia and dropsy appear to be the direct result of inflammation of kidney tissue, whereas in chronic Bright's disease they are probably more closely related to disturbance of circulation through the heart. At any rate the occurrence of uræmia and dropsy in a case of chronic Bright's disease should make us ask ourselves—Are these conditions dependent on circulatory failure or on acute nephritis, which has recently attacked already degenerated kidneys?

It must be admitted that in many cases of cardiac enlargement associated with dropsy and albuminuria, it is very difficult to say whether these phenomena depend on primary heart disease or on primary kidney disease. A history of shortness of breath preceding the dropsy would be in favour of original heart disease, also the presence of aortic or of diastolic or presystolic mitral murmurs. But a

systolic murmur at the apex might be due to cardiac dilatation, either from primary weakness of cardiac muscle, as in chronic alcoholism, or from muscle failure, following the hypertrophy due to chronic renal disease. In the latter case, too, the urine and the pulse might have all the characters present in cardiac disease. In such complicated cases there is perhaps only one sign which can be regarded as of diagnostic significance, namely, **albuminuric retinitis**. If well-marked signs of this condition are seen with the ophthalmoscope, then the presence of kidney degeneration can be pretty confidently asserted.

It is obvious, then, that the effects of kidney disease are often far-reaching, and may produce symptoms indicative of disturbance of the nervous, circulatory, digestive, or respiratory system, rather than symptoms indicating disease of the urinary tract. A recognition of this fact should lead us to examine the urine and the renal regions in all cases of obscure illness, or whenever vomiting, headache, giddiness, dyspnœa, or other prominent symptoms cannot be satisfactorily explained by an examination of the systems with disease of which they are usually connected.

Of symptoms pointing more directly to disorders of the kidneys, such as alterations in the frequency of micturition, pain along the urinary tract, &c. (see pp. 24–26), some facts relating to the latter subject may now be briefly considered.

When a patient complains of **pain** in the lower part of the back, it is more likely that he is suffering from lumbago than from kidney disease. At the same time a dull aching in the loins may be present in any form of kidney disease, although it is frequently overshadowed in the patient's mind by other symptoms, such as weakness, dropsy, or disturbances of micturition. Severe pain is exceptional in Bright's disease; it is often a prominent feature of suppurative nephritis, and may attend any variety of enlarged kidney.

When the pelvis and ureter are implicated, pains radiate down the side of the abdomen to the groin and testicle. Frequently they extend to the thighs, and sometimes even to the heel or sole of the foot. Paroxysmal attacks of **renal colic** may accompany the discharge of blood clots, hydatid cysts, or tubercular debris along the ureter, but are usually most marked and frequent as a result of the passage of a calculus along the ureter, or of disturbance of a calculus in the pelvis, or in one of the calyces of the kidney, as by shaking or jolting movements of the body.

In these cases the **testicle** is often very painful, swollen and tender on pressure. But let it be noted that irritation of the renal pelvis or ureter is not the only cause of pain referred to the testicle and inside of the thigh. Aneurysm of the abdominal aorta may lead to this pain

in the left testicle, disease of the vermiform appendix to a referred pain in the right testicle.

Moreover, it must not be forgotten that persons of a gouty diathesis may suffer from symptoms resembling those of renal calculus—pain and frequent micturition—simply as an effect of the passage of very acid urine, or of urine overcharged with uric acid. There may even be a trace of pus or blood in the urine, while in some cases patients suffer from testicular pain or soreness.

Further, it is of the greatest practical importance to know that symptoms pointing to an affection of the bladder and urethra, namely, pain, spasm, and irritation at the neck of the bladder and along the urethra, together with frequent micturition, may depend on **remote irritation**, as (1) from disease of the intestines, especially of the colon and rectum, or (2) from kidney disease. The close symptomatic relations existing between the bladder and kidneys are well summarised by Mr. Morris as follows:—

"Various functional disturbances and organic diseases of the kidneys are attended with so little if any pain referred to those organs, but with so much pain and irritation in the bladder and urethra, that the bladder or urethra, and not the kidneys, are considered to be the parts affected." "In some cases in which the symptoms excited by renal disease are all referred to the bladder and urethra, no organic lesion is found in the lower urinary organs even after prolonged illness; in other cases secondary organic lesions are produced in the bladder; and lastly, in some calculous and other diseases of the bladder most serious and extensive lesions may be present in the kidneys, although pain in the loins is not complained of, and the only malady indicated by the symptoms is referable to the bladder."

Scrofulous kidney may be particularly mentioned as an example where diagnosis is often difficult. Purulent urine and symptoms of vesical irritation in the absence of pain, tenderness, or palpable swelling in the renal regions, suggest disease of the bladder rather than of the kidney. But the bladder may be sound while the kidney is extensively diseased.

Errors in diagnosis are usually avoided by making careful examinations of the urine for several days in succession, by sounding the bladder, by a digital examination of the rectum, by careful palpation of the loins and abdomen, and finally by inquiries as to pain, the state of the digestive organs, and any other symptoms that may be present.

Anatomical Relations.—The kidneys are deeply seated in the abdominal cavity, on a level with the last dorsal and two or three upper lumbar vertebræ. Each rests from above downwards on the diaphragm, the quadratus lumborum and the psoas muscles. On the

front of the right kidney, uncovered by peritoneum, rest the duodenum and the commencement of the transverse colon; on the front of the left kidney lies the descending colon. The upper ends of the kidneys, capped by the suprarenal capsules, are nearer together than the lower, which reach nearly as low as the crest of the ilium. The upper portion of each kidney is covered by the eleventh and twelfth ribs, and partly by the complemental sac of the pleura, but it is below the edge of the lung. The upper border of the left kidney touches the spleen; that of the right kidney, the under surface of the liver.

The outer borders of the kidneys correspond to the edges of the

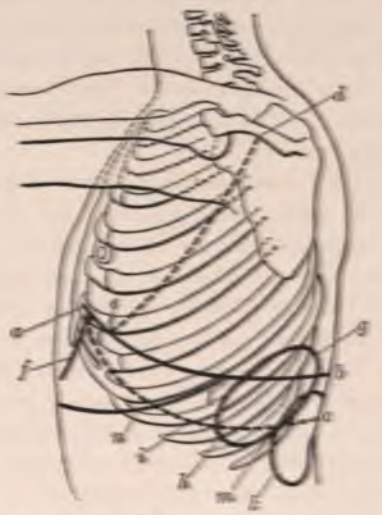


FIG. 183.—Relations of Spleen and Left Kidney (*Weil*): *g*, *h*, spleen; *k*, left kidney; *a*, *b*, lower margin of left lung; *a*, *c*, lower edge of pleural sac; *d*, *e*, fissure between lobes of lung; *f*, edge of left lobe of liver; *l*, angle between spleen and lung; *m*, angle between spleen and kidney; *n*, position of great curvature in moderate distension of the stomach.

erector spinæ, while the inner concave borders are close to the transverse processes of the vertebræ.

Inspection.—Only a great enlargement of the kidney produces a visible bulging of the abdominal wall in the lumbar region; distension of some of the superficial veins may then be seen, together with possibly a deficiency in the diaphragmatic movement on the affected side. A visible swelling on the posterior aspect of the lumbar region may also be produced by an extravasation of blood, or by a collection of pus round the kidney (perinephric abscess). In the latter case, the skin over the tumour is often red and œdematous.

Palpation.—The usual statement in the text-books that a kidney of normal size is accessible to palpation only in exceptional cases is, we

believe, an erroneous one. If the following method, as taught by Sir William Jenner, be carefully practised, the kidney may be felt in the large majority of healthy persons, and quite distinctly, as a rule, in children, and in women with lax abdominal walls.

Method.—One hand should be placed at the back of the patient below the last rib, and outside the lumbar muscles in the spinal groove; the other on the front of the abdomen, just over the hand behind. The hand in front should then be depressed—the patient's attention being diverted, and his muscles relaxed as much as possible—while the hand behind is tilted forward, when the kidney will be brought well under the touch of the two sets of fingers.

The loins should also be examined (1) when the patient is standing up, and (2) when he is on his hands and knees.

Displacement.—A "**movable**" kidney is one that is readily displaced from its normal position, and which may be felt as a smooth, slippery tumour in the umbilical or other region of the abdomen. The condition may be congenital or acquired. Congenital mobility, called also "**floating** kidney," is due to the presence of a true mesentery, the organ being partially or completely surrounded by peritoneum. Acquired mobility is much more common; it occurs chiefly in women who have had repeated pregnancies, and the right kidney is much more frequently affected than the left. It may be pushed in almost any direction, but more easily upwards towards the thorax. Pressure over the reniform tumour is usually painful, and causes a sickening sensation; pulsation from the renal artery is sometimes perceptible. The tumour alters its position with that of the body; it is liable to become enlarged, and more tender to palpation during the menstrual period; sometimes the enlargement is due to hydronephrosis, rarely to cancer, the nodules of which may be palpable. A movable kidney, or one temporarily displaced by some diseased condition in the vicinity, may become permanently fixed in its new position. Whenever one of the kidneys occupies an abnormal position, the posterior lumbar region corresponding to it will often appear less full and feel less resistant than on the opposite side.

Sometimes the connecting isthmus of a "**horse-shoe kidney**" is palpable as a firm band transversely placed across the lower part of the abdomen.

Enlargements.—A renal tumour occupies at first the anterior part of the lumbar region; it then extends upwards into the hypochondrium, backwards towards the spine, forwards to the umbilicus, and downwards to the iliac crest. When not very large, and pressing, as it then does chiefly anteriorly, it may closely resemble the spleen; but as a rule, though by no means invariably, a hollow is felt between the upper part of a renal tumour and the costal margin, whereas an enlarged

spleen passes beneath the costal margin, and there is never any perceptible line of separation between the two. In front, too, of a renal tumour, the colon is commonly felt as a soft or yielding structure, sometimes as a thickened cord; and separating as it does one portion of the renal enlargement from another portion, it gives the idea that there are two tumours instead of one. Inflation of the colon is sometimes an aid to diagnosis. The presence of intestine in front of an abdominal tumour, although testifying to its renal origin, cannot be regarded as an absolutely constant phenomenon.

The anterior margin of an enlarged kidney is rounded, whereas that of an enlarged spleen is sharp and notched. Every part of the outline of a renal tumour that can be felt is rounded; there are no sharp edges. A renal tumour enlarges in front, it fills up the hollow of the loin, and but rarely causes enlargement behind, whereas abscesses and other lesions in the renal region may cause considerable projection posteriorly. A distended gall bladder may simulate a fluctuating renal tumour; but the latter can be traced back to the spine, whereas the former is superficial, and pyriform in shape.

The following are the chief lesions which give rise to palpable enlargements of the kidney:—

Pyelitis and pyonephrosis.
Hydronephrosis.
Tubercle, cancer, sarcoma.
Hypertrophy.
Cysts, simple, hydatid, or dermoid (very rare); "cystic disease."
Infarcts and abscesses.

In young children the commonest and largest tumours are due to congenital hydronephrosis and sarcoma. A sacculated tubercular kidney, although not uncommon, is less frequently palpable than the two lesions just mentioned.

In hydronephrosis and in hydatid cyst, the swelling is soft, elastic and fluctuating. In pyonephrosis fluctuation is often obscure, and indeed a tumour in the flank is usually a late phenomenon. In these diseases variations in size are liable to occur in accordance with the presence or absence of any discharge of fluid into the ureters. Such changes in size are especially characteristic of hydronephrosis, where sometimes a sudden disappearance of the lumbar swelling is coincident with the passage of a large quantity of urine. A feeling of doubtful fluctuation is occasionally present in cancer or sarcoma, but more commonly the tumour is firm and fixed, and presents lobulations or nodules of varying degrees of hardness.

Percussion.—This is a much less reliable method of investigation than palpation. Normally when a person lies prone with the abdomen

well supported, an area of dulness is obtained posteriorly, which passes upwards into the splenic dulness on the left, and into the hepatic on the right side, but is limited below and on the outer side by the tympanitic note of the colon. When a kidney is displaced from its normal site, a tympanitic note is commonly yielded by percussion of the corresponding side. An enlargement of the kidney, not great enough to be accurately palpated, may sometimes be detected, and its size roughly estimated, by means of percussion. In front of a large renal tumour, a band of resonance, often a tubular note, is elicited by gentle percussion, unless the colon is filled with solid material, or is unduly compressed.

CHAPTER X.

EXAMINATION OF THE URINE.

VARIATIONS IN THE QUANTITY OF THE URINE.

In health the average daily discharge ranges between 40 and 50 fluid ounces, but in different individuals, or in the same person under different conditions, considerable oscillations may occur. More than 80 or 90 or less than 18 oz. is usually to be regarded as pathological.

The new-born infant passes from 7–12 oz., and the quantity secreted per hour is much more regular than in the adult. The quantity of urine is increased after meals; it is diminished during fasting and during sleep.

In Disease the Quantity is Increased (*polyuria*) :—

1. Most markedly in diabetes mellitus and diabetes insipidus, when from 100–400 oz.—that is, eight times the normal quantity—may be passed.

2. Whenever there is an increase of the blood pressure, as in granular kidney, and in cases where compensation for cardiac lesions is beginning to be established.

3. In certain stages of lardaceous kidney.

4. At the crisis of, or during convalescence from, some febrile diseases; this is especially noticeable in typhus.

5. When there is active absorption of dropsical collections, or after the removal of some of the fluid.

6. In certain cases of injury or of disease of the nervous system. Thus polyuria often follows emotional disturbance, attacks of epilepsy or of hysteria, and may occur shortly after hæmorrhage into the brain.

7. After the administration of certain drugs, as digitalis, the salicylates, calomel and the salts of potash.

The Quantity is Diminished (*oliguria*) **or Entirely Suppressed:—**

1. In congestion and in acute inflammation of the kidney; also in the terminal stages of all forms of Bright's disease.

2. From mechanical impediment to the flow of urine. (*a.*) When diminished secretion is caused by an **obstruction** in the ureter or pelvis of the kidney, any urine passed is pale, watery, and devoid of albumin and casts. (*b.*) When the diminished secretion is **non-obstructive**, and results from organic disease or from some neurotic disturbance of the renal functions, the urine is high-coloured and concentrated; in such cases disease of the renal tissue may be indicated by the presence of albumin and casts.

In a case of hysteria reported by Charcot, no urine was secreted for eleven days.

3. When there is a great loss of water from other parts of the body —as in all fevers, in acute rheumatism, in the collapse period of cholera, during the obstinate vomiting which sometimes occurs in hysteria, and in obstruction of the upper part of the bowel; also when there is profuse diarrhœa.

4. In cirrhosis of the liver.

VARIATIONS IN THE COLOUR OF THE URINE.

The tint in health is brownish-yellow, varying from a straw yellow to a golden amber. The depth of colour is dependent on the degree of concentration; thus urine passed in the early morning is more concentrated, and therefore higher coloured than that passed later in the day; after drinking much, the urine may be almost colourless. In disease there is not always a relation between the intensity of colour and the quantity of the urine; very dark-coloured urines may be met with when the excretion is abundant, and *vice versâ*.

Pale urine occurs in diabetes mellitus and insipidus, in chronic Bright's disease, in hysteria, in anæmic conditions, and during convalescence from severe disease. It suggests the absence of pyrexia.

Whitish urine, in which the colour is due to the presence of white urates, is frequently passed by children who are out of health from any cause.

Yellowish and milky urine may result from the presence of (1) fatty drops floating in the urine, as in chyluria, or (2) suspended pus corpuscles, as in pyelitis.

Yellow, green, to black urine indicates the presence of bile pigment,

and is therefore of frequent occurrence in cases of jaundice. It also occurs in cases of carbolic acid poisoning; after doses of salol, or of salicylate of soda; and in consequence of hæmorrhage into the kidneys, the hæmoglobin having become decomposed. Urine containing melanin becomes black on standing.

Dark yellow to brownish-red urines are seen in certain pyrexial diseases, as rheumatism, pneumonia, in passive congestion of the kidneys, after much diarrhœa, and in liver diseases. Urine rich in indican or in pyrocatechin has usually a deep brown colour.

Reddish urines, varying in tint from pale pink to a bright ruby red, suggest the presence of blood.

The urine may also be considerably altered in colour from the admixture of certain drugs, such as rhubarb, senna, or santonin.

VARIATIONS IN THE ODOUR OF THE URINE.

Healthy urine has a characteristic aromatic smell which varies in strength. Urine emits a fœtid and ammoniacal odour when it contains ammonium carbonate as a result of fermentation of urea. Such ammoniacal fermentation takes place in any urine after standing for some time; if it takes place in the bladder, the urine is ammoniacal when voided. In the latter case it may even have a putrid or a sulphurous odour. The presence of sugar in the urine is often attended by a sweet smell; the presence of acetone or of aceto-acetic acid by a smell like that of chloroform. Turpentine taken internally gives an odour of sweet violets to the urine. Cubebs, copaiba, valerian and other drugs, also onions and other vegetables, communicate odours to the urine.

VARIATION IN THE CONSISTENCE OF THE URINE.

The urine in diabetes insipidus, and in some cases of hysteria, is as clear as pure water. An ammoniacal purulent urine is viscid and ropy, and is not readily poured from vessel to vessel. The consistence of urine is also increased by the presence of molecular fat as in chyluria; when there is much albumin the splash on pouring is softer, and more froth is formed than in health. Fibrinuria, a rare condition, is recognised by the spontaneous coagulation of the urine.

VARIATION IN THE TRANSLUCENCY OF THE URINE.

Healthy urine is perfectly clear, except for the delicate cloud of mucus which appears after standing. Temporary opacities are produced by an excess of the earthy phosphates or of the amorphous

urates, but their subsidence leaves the supernatant fluid transparent; occasionally, however, it remains somewhat opaque. Opacity of freshly voided urine is usually due to pus; sometimes to phosphates; and rarely to the presence of fat as in chyluria—the urine then looks cloudy and milky.

VARIATIONS IN THE SPECIFIC GRAVITY OF THE URINE.

The specific gravity is conveniently estimated by the urinometer. The instrument should be large, with the divisions of the scale wide apart. Urinometers are usually graduated at a temperature of 15.5 C.; if the urine when examined is of a higher temperature, one degree of sp. gr. must be subtracted from that recorded by the instrument for every three degrees of temperature (C.). The instrument should float freely, without touching the side or the bottom of the vessel. In reading it the eye must be lowered to the level of the surface of the fluid, and the reading taken there and not from the top of the curve formed by the urine round the stem of the urinometer. A control estimation is easily made by depressing the instrument in the urine, again allowing it to come to rest, and again reading.

The specific gravity of healthy urine varies greatly during the twenty-four hours; after much drinking on an empty stomach it may be 1005 or even lower, after profuse perspiration it may reach 1028 or higher. The average healthy variation, however, is 1015–1025; in infants it is only 1003–1006.

In health the sp. gr. is proportional to the degree of colour. We expect a low sp. gr. in pale urines, and generally this holds in disease, so that the conditions mentioned as increasing the quantity of the urine, and so diminishing its colour, also lower its sp. gr.; those which diminish its quantity, and so heighten the colour, raise the sp. gr. Thus the scanty urine of uncompensated mitral disease gives a high sp. gr., the pale profuse urine of granular kidney a low sp. gr. Diabetes mellitus forms an important exception to this rule. Here the quantity of urine passed is very large, its colour pale, but the sp. gr. is very high, and may reach 1060. An excess of urea or of albumin will also sometimes considerably raise the sp. gr.

An approximate **estimation of the solid matters** passed, in grains, may be made by multiplying the last two figures of the sp. gr. by the number of ounces of urine passed in the twenty-four hours. Thus a healthy adult of average weight will pass about 50 oz. of urine of sp. gr. 1020, then $20 \times 50 = 1000$ grs. or about 4 per cent. of solids; but one-fifth above or below this should be allowed for healthy variations. The qualifying influence of weight should always be considered,

for in an adult each pound of body weight yields from 7 to 8 grs. of urinary solids. It is to be observed that children under seven years excrete, in proportion to their weight, nearly twice the amount of solids that adults do.

A sudden lowering of the sp. gr. in some diseases sometimes precedes a fatal turn. A fall in sp. gr., without any material alteration in the quantity of urine, suggests inability on the part of the kidneys to excrete sufficient urea and salts. Such a condition of renal inadequacy may precede the development of renal cirrhosis; when it occurs during the course of Bright's disease, the sp. gr. of the urine falling somewhat suddenly without alteration in the bulk of the urine, it may be the forerunner of a uræmic attack, even in the absence of other premonitory symptoms.

VARIATIONS IN THE REACTION OF THE URINE.

The reaction is ascertained by means of blue and red litmus papers; the latter not only detect alkalinity, but distinguish between that due to fixed alkali (potash or soda) and that produced by volatile alkali (ammonia); thus, if the blue colour persists after the paper has been completely dried, the alkali is fixed; if it vanishes, the original red colour being restored, the alkalinity is due to ammonia. Degrees of acidity or alkalinity are roughly estimated by the amount of changes of colour. When the reaction is neutral, neither paper changes colour. Violet-tinted papers with glazed surface are the most delicate; they are turned red by acid and blue by alkaline fluids. The acidity of the urine may be estimated quantitatively by means of a standard solution of soda.

Healthy urine is generally acid, but the reaction fluctuates much during the twenty-four hours; it is more acid before meals and during sleep: it is less acid after meals, when the urine may be neutral or even alkaline.

The alkalescence following food is due to fixed alkali; it is only observed when the urine is examined at hourly intervals, for otherwise the urine, though alkaline as it leaves the kidney after meals, mixes in the bladder with acid urine secreted before and after the alkaline tide, and is thus rendered acid.

Influence of Medicines.—The urine is easily made alkaline by the administration of alkaline substances, but acids, excepting benzoic acid, have but little power in raising the acidity of the urine, or in turning acid a urine habitually alkaline.

Influence of Disease.—**Alkalinity from Fixed Alkali** occurs in cases of debility and anæmia, in chronic vomiting and in phthisis. The

condition is generally fugitive, but it may persist for weeks. Such urine is secreted alkaline by the kidneys, and exhibits little or no deposit.

Ammoniacal urine, on the other hand, is very rarely furnished as such by the kidneys, but is derived from decomposition of urea in the lower urinary passages; it shows a sediment, and is highly irritating to the mucous membrane over which it travels. The change is due to the action of the *micrococcus ureæ*, and probably also sometimes to other organisms. The commonest cause of ammoniacal urine is inflammation of the bladder; any condition, such as stricture of the urethra or paralysis of the bladder, which interferes with complete emptying of the viscus favours the production of ammoniacal urine.

Increased Acidity of the urine occurs sometimes in febrile conditions, in diabetes, leukæmia and scurvy.

CHEMICAL EXAMINATION OF THE URINE.

Albuminoid bodies not present in health are divisible by heat into two classes:—

A. Those coagulated by heat, namely, serum albumin and globulin; these are proteids natural to the blood.

B. Those not coagulated by heat, or derived proteids, viz.:—(1.) Albuminates consisting of albumin in combination with an acid or an alkali. (2.) Peptones. (3.) The albumoses.

Serum Albumin.—To determine in any given specimen of urine the presence or absence of serum albumin is, from a clinical point of view, of the highest importance.

Of the numerous tests which have been used for the detection of albumin, by far the best are the boiling test and the nitric acid test.

The Boiling Test.—In applying this test, the proper acidulation of the urine is of great importance; this may be attended to either before or after boiling.

(A.) **Acidulation after boiling.**—A test tube two-thirds full of the urine to be examined is held by its lower end, while the upper layer is heated in the flame of a lamp till it begins to boil.

The boiled portion of fluid should now be carefully compared with the cool portion in the lower part of the test tube by holding it against a black surface. If any cloudiness appear, add a drop or two of dilute nitric or acetic acid. Then, if the turbidity remains, it is due either to serum albumin or to globulin; if it disappears, to earthy phosphates.

Precautions.—1. The urine must be clear; if turbid from the presence of amorphous urates, it may be quickly cleared by gently warming the test tube. If alkaline and turbid from phosphates, a drop of acetic

acid should be added before boiling, otherwise the albumin will not be precipitated. If the urine is cloudy from an organic source—mucus, pus, blood, &c.—it should be filtered.

2. The urine must not be too acid, for if so the serum albumin may be converted into acid albumin, which does not coagulate with heat. An unwashed test tube containing a drop of nitric acid is the commonest source of error; even a great natural acidity of the urine has been known to prevent the precipitation of albumin by heat. The addition of a little liquor potassæ, however, speedily converts the acid albumin into serum albumin, when coagulation will occur with heat.

3. The urine must not be too alkaline, otherwise alkali albumin is formed, which, like acid albumin, is not coagulable by heat.

(B.) **Acidulation before boiling.**—This is a more delicate mode of applying the heat test; for if an albuminous urine of normally acid reaction is boiled, and after the addition of a drop of acetic acid is filtered, the filtrate may be shown by means of other tests (such as the picric acid or the mercuric iodide test) to contain a small quantity of albumin. But if, before boiling, the urine is acidified *to the proper degree*, and then boiled and filtered, no albumin, or only the merest trace, can be detected. The delicacy of the test is dependent on the *proper degree* of acidulation, which demands great care, for the addition of a slight excess of acetic acid may prevent the precipitation of albumin.

Sir William Roberts, who introduced this method, recommends the addition of one drop of B.P. acetic acid to ʒiii (10 cc.) of urine. On boiling the upper portion of this fluid, a slight opalescence reveals the most minute quantity of albumin. "If the urine be alkaline, it should be carefully neutralised by adding successive drops of acetic acid until the litmus paper shows a distinct but slight acidity, and then the final single drop of acid is added before boiling. Even if the urine possesses its natural acidity, it is better to add a drop of acid if you want to bring out the maximum sensitiveness of the boiling test. When performed with these precautions, the boiling test is the most sensitive and the most reliable of all albumin tests."

The Nitric Acid Test.—The addition of nitric acid to the cold urine, so that it forms a distinct and separate layer below it, is one of the most convenient, simple and delicate means for the detection of albumin. This "contact method" is more readily carried out by putting the acid into the test tube first than by trickling it along the side of the tube through the urine to form a layer below. The bottom or narrow part of the test tube should be first filled with the strong acid, then the lighter urine should be poured very slowly down the side of the tube,

which should be held almost horizontally. With care the urine may be poured directly out of a bottle or containing glass, but mixing of the two liquids is more certainly avoided by using a pipette, by means of which the urine can be dropped slowly on the side of the tube held nearly horizontally. The presence of albumin is shown by a cloud or a ring of haziness at the junction of the two fluids; below this opalescent zone of albumin is the colourless nitric acid, above it is the unaltered urine. This test precipitates serum albumin, globulin and albumoses, but does not coagulate peptones.

Precautions.—(1.) If the quantity of albumin is very small, many minutes may elapse before an opalescent zone appears; when, therefore, the immediate reaction is negative, the tube should be set aside for twenty to thirty minutes, and then re-examined.

(2.) Nitric acid may precipitate amorphous urates; the precipitate, however, appears first near the surface of the urine, and not, like albumin, immediately above the acid; also, it quickly disappears on warming the test tube.

(3.) Turbidity of the urine interferes with the delicacy of the test. When the turbidity is due to urates, the commonest cause, slightly warming the test tube will clear the urine, which should then be immediately trickled down on to the surface of the acid.

(4.) In urine loaded with urea, nitric acid causes precipitation of nitrate of urea. But, being crystalline, the precipitate has a very different appearance from coagulated albumin; it is also speedily dissolved by gentle heat.

(5.) Patients who are taking cubebs or copaiba often pass opalescent urine, and the opalescence is increased by contact with nitric acid. Heat lessens the haziness, and the presence of these drugs is also indicated by their characteristic odour.

(6.) Mingling of the urine and acid may prevent coagulation of the albumin; hence the great importance of avoiding any sudden fall of the urine on to the acid.

(7.) The presence of mucin may be shown by a diffused haze, or sometimes even by a distinct zone, but this is seen in the middle or upper part of the column of urine quite away from the level of the nitric acid.

From the above account it is seen that most opacities produced by other than albuminous bodies quickly disappear on the application of heat.

Magnesio-Nitric Test.—This is a modification of the nitric acid test. The liquid used consists of a mixture of one volume of strong nitric acid with five volumes of a saturated solution of sulphate of magnesia. It is a clear solution, and by the contact method is an excellent test

for albumin. It does not fume, and is much less destructive to the skin and clothes than the undiluted acid. It is as delicate, if not a more delicate test for albumin than nitric acid alone. It more readily precipitates mucin, and the mucin ring is nearer the test solution than when the undiluted acid is used. In applying this test, the intermingling of the reagent with the urine must be carefully avoided.

Picric Acid Test.—A saturated solution of picric acid, when dropped into an albuminous urine, causes an opacity; the picric acid should be in excess, and if the urine is alkaline a stronger acid, such as citric or dilute acetic acid, is also necessary. The "contact method" is more delicate. The urine should be first acidulated with citric acid, and then the picric acid solution floated by means of a pipette on to its surface. Opacity at the juncture of the two liquids may be due to serum albumin, globulin, peptones, albumoses, an alkaloid (especially quinine) or to urates. If the precipitate be caused by any of the four last-named bodies, heat will dispel it, but an albumin or globulin precipitate is permanent.

One of the greatest sources of confusion with the above reagents is mucin, which, except by nitric acid, is thrown down as a zone at the junction of the two fluids; the haze of mucin produced by nitric acid is well above the line of contact, and so is more easily distinguished from the lower, white, albuminous cloud.

Salicyl-Sulphonic Acid Test.—If a couple of drops of a saturated solution of salicyl-sulphonic acid are added to a small quantity of albuminous urine, and the mixture is shaken, a more or less marked opalescence occurs either at once or after standing a few minutes. If the opalescence does not appear until a few minutes have elapsed, only a trace of albumin is present. Serum albumin, globulin, albumose and mucin are precipitated by this reagent; if the urine is subsequently heated, the precipitates due to albumin and globulin are coagulated and are permanent; the precipitates due to albumose and mucin are dissolved by heat, and are reprecipitated on cooling. Peptones are not precipitated unless the urine is previously saturated with ammonium sulphate. To obtain a delicate reaction with salicyl-sulphonic acid, the urine to be tested must be free from turbidity. With alkaline urine the reagent must be added until marked acidity is produced.

The Quantitative Estimation of Albumin is roughly made by boiling some urine in a test tube, adding to it a drop or two of acetic acid, and then setting it aside for a few hours; the depth of the deposit of coagulated albumin is then compared with the total depth of the urine. If the height to which the albumin reaches is equal to one-fifth of the column of urine, there is said to be one-fifth of albumin. If the

quantity of albumin is too small to enable a quantitative estimation to be made, it is spoken of as a "trace." It has been found that if the urine completely solidifies on boiling, the quantity of albumin is from 3 to 5 per cent. (the quantity in blood serum being 5 per cent.). If half the column is represented by coagulated albumin, there is from 1 to 2 per cent. If only a "trace," about 0.05 per cent. The total daily loss in grains is obtained by multiplying the percentage by 4.36, and then by the number of ounces of urine passed in the twenty-four hours. Thus, if the proportion of albumin equals .5 per cent., and the amount of urine passed in twenty-four hours equals 60 oz., then $.5 \times 4.36 \times 60 = 130.8$ grs. is the amount of albumin passed in that period. The sample of urine should be taken from the total amount passed during the twenty-four hours. If urates have deposited, they must be filtered off before boiling the urine. The result obtained is only an approximate one.

FIG. 184.—Esbach's Albuminimeter, ½.

Quantitative Estimation of Albumin by Esbach's Process is a more satisfactory method than the preceding. According to this method the albumin present in the urine undergoing examination is coagulated by a chemical reagent instead of by boiling. The reagent used is prepared by dissolving 10 grains of pure picric acid and 20 grains of pure citric acid in 900 cc. of hot water. When the solution is cool, sufficient water is added to make 1000 cc. or 1 litre.

The urine to be tested is poured into a specially graduated tube (see Fig. 184) up to the mark "U," and sufficient of the reagent added to reach the mark "R." The tube is then closed by the stopper, and reversed two or three times without shaking, after which it is allowed to stand upright for twenty-four hours, when the height of the sediment is read off by the scale at the lower end of the tube. The figures express the number of grammes of proteids (chiefly serum albumin and globulin) which are contained in one litre of urine. The results yielded by this process are sufficiently accurate for clinical purposes, the object being to trace the progress of a case by periodic estimation of the daily amount of albumin excreted rather than to obtain an absolutely accurate determination from a single specimen of urine.

Precautions.—The most accurate results are obtained by attention to two points.

1. The urine must be fresh and acid.
2. If the urine is of high specific gravity, or if it contains a large

quantity of albumin it should be diluted; the amount of dilution being allowed for when estimating the number of grammes in a litre.

In some instances, after the tube has stood for the usual time, part of the coagulated albumin remains floating in the upper stratum of the fluid; in such cases the tube should be closed, well shaken, and allowed to stand for twenty-four hours longer.

The Clinical Significance of Serum Albumin in Urine.—In investigating a case of albuminuria the observer should remember that the causes are often complex, and that the presence of albumin in urine is merely a symptom which must be weighed in connection with all other symptoms present, before its significance can be properly estimated, and before the all-important question as to the presence or absence of organic disease of the kidneys can be decided. The daily quantity of albumin should be ascertained, and also whether its presence is constant or intermittent (see *Cyclic Albuminuria*), the investigator noting in the latter case the influence of food and of exercise, and remembering that the maximum daily amount of albumin is usually to be found in the urine passed some two hours after breakfast. The amount of albumin should be considered in relation to the specific gravity and the colour of the urine, and to the microscopic appearance of any deposit. The possible existence of symptoms pointing to disease of other organs than the kidneys has also to be carefully considered.

If the urine contains blood or pus, it is often a nice point to decide whether the amount of albumin is out of proportion to that which would be derived from the blood or the pus present: daily examinations will probably clear up any doubt, since in one set of cases variations in the quantity of blood or pus will be accompanied by variations in the quantity of albumin, but in another set of cases the amount of albumin will show less change, thus affording evidence of an independent origin of much of the albumin which is present. The microscopic nature of the organic deposit, and a general consideration of all the symptoms presented by the patient will in most cases afford a clear explanation of the sources of the albumin detected in the urine.

The Chief Causes of Albuminuria are:—1. Acute and chronic Bright's disease of the kidneys.

2. Obstruction of the general venous circulation as the result of heart disease, emphysema, &c.

3. Acute febrile and inflammatory diseases such as scarlet fever, pneumonia, peritonitis.

4. Pregnancy and the puerperal state.

5. Obstruction of the ureters.

6. Certain nervous affections, especially cerebral hæmorrhage, concussion of the brain, epilepsy, tetanus, delirium tremens.

7. Various chronic diseases such as anæmia, leukæmia, exophthalmic goitre.

8. Poisoning by lead or other substances.

9. Certain functional disorders, as dyspepsia.

10. Disease of some part of the genito-urinary tract—the albumin being derived from pus or blood mingling with the urine—as in pyelitis, cystitis, leucorrhœa, or gonorrhœa.

Cyclic Albuminuria.—It sometimes happens that a small quantity of albumin is found in the urine of persons who appear to be quite healthy. In some cases it can only be detected in urine passed after breakfast, or after muscular exertion; in others it regularly appears in the urine at certain periods of the day, and is absent at other times. The exact significance of this condition is not fully understood; it is probably related to changes in the renal circulation. Owing to its greater frequency at the period of puberty than at other ages, it has been called the "*albuminuria of adolescents.*"

Globulin is chemically allied to, and nearly always found in association with, serum albumin. It is precipitated by heat and other albumin precipitants. Like serum albumin, it is soluble in a saline fluid such as urine, but it differs from serum albumin in its insolubility in pure water. On this property Sir William Roberts brought forward the first of the following tests.

Tests.—1. Fill a glass cylinder with *distilled* water and drop into it some albuminous urine. If globulin be present, each drop will be followed by a milky train, and if enough urine be added the water becomes quite milky. The milkiness is dissipated by a drop of acetic acid or of liquor potassæ.

2. Take some urine in a test tube and make it neutral or very faintly alkaline with liquor potassæ, then pour a saturated solution of magnesium sulphate down the side of the test tube, and the presence of globulin is indicated by a well-defined narrow white ring at the junction of the two fluids. All the globulin may be precipitated by completely saturating the neutralised urine with magnesium sulphate; if this mixture be now thrown on to a filter, a clear liquid will come through, which on acidulation will give the tests for serum albumin, should this body be present.

The relative quantity of globulin and serum albumin in the urine may be estimated by means of Esbach's process. The total quantity of proteids is first ascertained by the method already described (see p. 316). Another portion of the urine is rendered faintly alkaline and the globulin precipitated by saturation with magnesium sulphate, and the mixture filtered. The filtrate contains any serum albumin present, and its amount is determined by Esbach's method; five or six days

should elapse before the tube is read off. The difference between the reading yielded by the last experiment and that given by the total quantity of proteids represents the amount of serum globulin.

Globulin, which probably never occurs in the urine except in conjunction with serum albumin, may often be detected in acute nephritis, and in the various forms of Bright's disease; also when there is catarrh of the bladder.

Peptones in the urine are not precipitated by heat, nitric acid, or ferrocyanide of potassium, but are thrown down by potassic mercuric iodide, by tannic acid and by picric acid; and they turn a rosy red colour in the presence of a weak alkaline solution of copper sulphate—the biuret reaction.

Tests.—1. **The Copper Sulphate Colour Reaction.**—(*a.*) Add to the suspected urine an equal quantity of liquor potassæ and one drop of a weak solution of copper sulphate. If albumin is present, the blue solution turns violet; if peptones, pink; if both albumin and peptones are present, a reddish-purple colour appears. Or (*b.*) float some of the suspected urine on to the surface of a little Fehling's solution. At the point of contact a zone of phosphates forms, and if peptones are present, the urine above turns a rosy or pink colour; if albumin is present as well as peptones, the colour will be mauve; with albumin only, it will be purple, while normal urine turns a greenish tint.

2. **The Picric Acid Test.**—This can only be applied when the urine is free from albumin, globulin and albumoses. These proteids may be removed by rendering the urine faintly acid with acetic acid, then adding powdered sulphate of ammonium, and agitating until complete saturation occurs. On filtering, the filtrate, if peptones are present, will give a cloud with a saturated solution of picric acid or with Esbach's solution; the cloud disappears with heat, but returns again on cooling.

Clinical Significance of Peptonuria.—The presence of peptones in the urine must always be regarded as a morbid phenomenon. They are often found in large quantities when albumin is absent, but albuminuria frequently exists without peptonuria.

Peptones have been detected in many diseases: during the course of several of the acute specific fevers, in rheumatic fever, croupous pneumonia, phthisis, and in cancer of the digestive tract. V. Jaksch considers that peptonuria is most commonly associated with such processes as are characterised by the collection and subsequent destruction of leucocytes, and that it is often a valuable diagnostic sign of the presence of suppuration in some part of the body. "Thus when peptone is found in the urine in the course of pneumonia, it indicates that the stage of softening has begun. Again, in connection with abdominal tumours or pleuritic effusion, it shows their purulent character; and

in purulent meningitis its manifestation varies with the severity of the disease."

It is important to know that peptonuria may be merely the result of the decomposition of albumin in the urinary passages, and therefore that, in any case of peptonuria, careful clinical investigation is necessary to determine whether the peptones are derived from the circulation, or whether they are the result of local action on some albuminous substance.

Albumoses.—These bodies, formed during peptic digestion, are to be regarded as being intermediate between globulin and albumin on the one hand, and peptone on the other. They are precipitated by acetic acid and ferrocyanide of potassium; give a pink colour (like peptones) with copper sulphate and potash, and yield a characteristic reaction with nitric acid.

Test.—Remove any albumin or globulin by acidifying the urine with acetic acid, boiling and filtering. Pour some of the filtrate on to the surface of a little cold nitric acid; if any albumose is present, a white cloud forms at the junction of the two fluids; the cloud dissolves on heating, but reappears on cooling.

Mucus and Pus.—Healthy urine after standing for some time shows a faint cloud of mucus floating near to the bottom of the vessel. Mucus itself is transparent, but is rendered visible by the presence of mucous and epithelial cells; these are very scanty in strictly healthy urine, and therefore the cloud is very delicate. When from any irritation of the genito-urinary tract the quantity of mucus and of the accompanying cellular elements is much increased, opaque thick clouds are seen, which pervade the whole urine and give it quite a glairy character. Between mucus and pus there is no abrupt line; the latter is usually present when the former is very abundant. When there is much pus, it falls as a thick yellowish-white creamy sediment; when the urine is acid, the deposit is loose; when alkaline, it takes the form of a gelatinous mass, which adheres to the bottom of the vessel, and can be drawn out into viscid strings.

Sources of Pus.—*Gonorrhœa* is the commonest cause in men, *leucorrhœa* in women. In the former case, more pus will be passed during the first half of micturition than during the second. In leucorrhœa the microscope will reveal much scaly epithelium from the vagina. In both cases the quantity of pus will be small.

In *cystitis* the urine is usually ammoniacal, and hence the pus forms a ropy mass; much mucus, too, is commonly present.

In *pyelitis* the urine is commonly, but not always, acid in reaction: there is not much mucus, and so the pus deposits more completely; there is not more pus during one part of micturition than another,

whereas in cystitis there is rather more pus passed during the second part of micturition.

Abscesses bursting into any part of the urinary tract will throw quantities of pure pus into the urine; the flow of pus is apt to be intermittent.

In disease of the prostate, and in gonorrhœa and gleet, fine threads are often seen in the urine, which microscopically are seen to be little plugs made up of aggregations of pus cells.

Tests for Mucin.—Mucin is precipitated by many of the tests for albumin, for which it is very liable to be mistaken. It is distinguished, however, by its non-coagulability by heat, and by its reaction with citric or acetic acid. Fill the bottom of a test-tube with a saturated solution of citric acid (or some acetic acid thickened by the addition of one-third its bulk of glycerine), pour some of the urine gently down the side of the test-tube; if mucin is present, a cloud forms just above the point of contact of the two liquids.

Tests for Pus.—1. Pour off the supernatant urine, and add liquor potassæ or liquor ammoniæ to the deposit, when, if it contains much pus, a viscid ropy gelatinous mass is formed; shaking and warming the test-tube aid the precipitation.

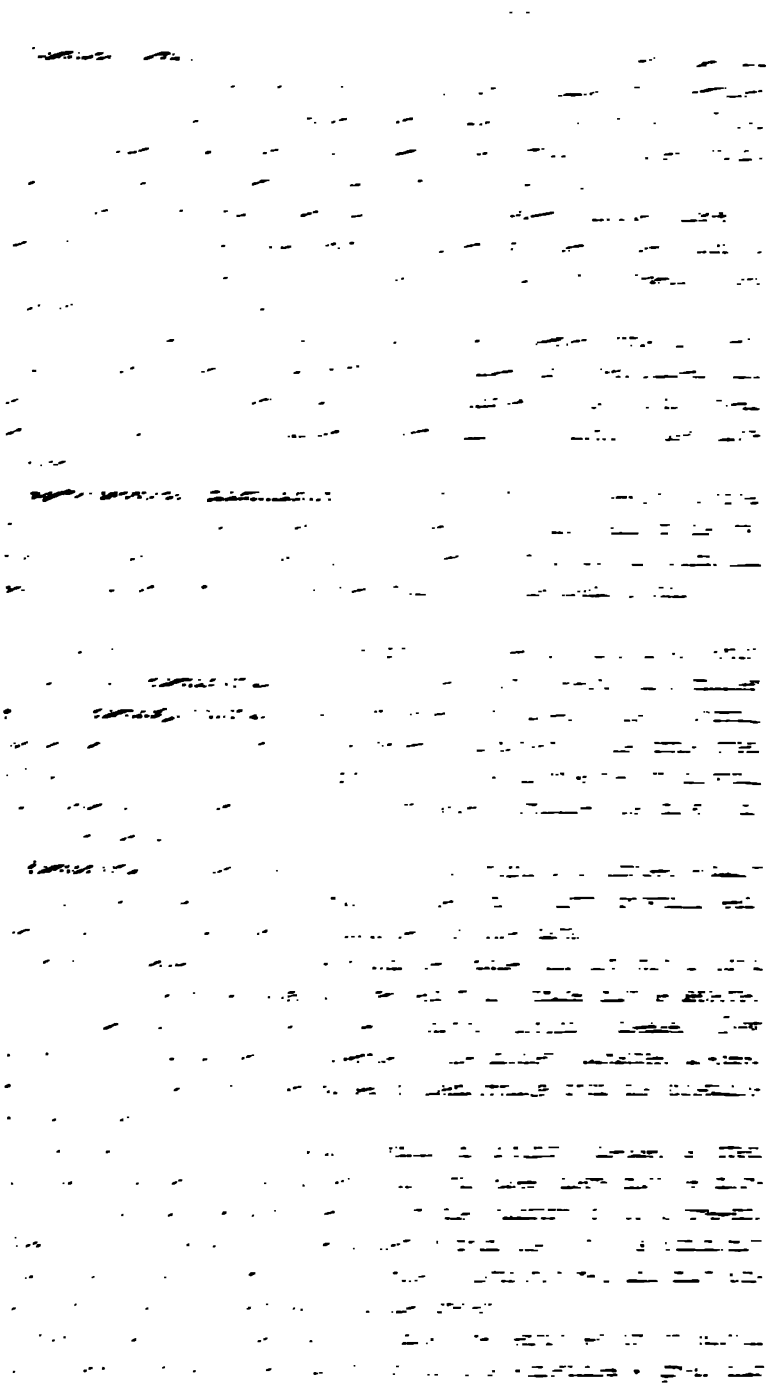
2. The nitric acid test shows an albuminous ring, but, as a rule, there is a mere haziness at the line of contact if pus only be present; a more decided, thicker, white zone is evidence of renal disease, and consequently casts should be carefully sought for.

3. Mercuric chloride gives a precipitate with pyin, but not with pure mucin, and thus the two may be distinguished.

Chyluria.—Exceptionally, the elements which constitute chyle may be present in the urine. This condition is probably always due to direct communication existing between the lacteals and the urinary passages. It mostly occurs in the tropics, and is there caused by a parasitic worm, the *filaria sanguinis hominis*, which breeds in the lymphatics, and burrows into the substance of the kidneys. Chylous urine is milky when passed, and usually coagulates on standing. Partial coagulation may occur in the bladder, the urine when passed containing shreds and clotted masses.

Blood.—A very small quantity of blood impairs the transparency of urine, and gives it a distinctive colour; a bright red if the urine is alkaline, or if the quantity of blood is more than one-fifth per cent.; a pale brown or a smoky tint if the urine is acid, or if only a trace of blood is present.

In addition to the altered colour of the urine, the presence of blood may be determined by chemical, microscopical and spectroscopical examination.



the urine coagulates spontaneously. Often more blood is passed with the last than with the first portions of the urine; commonly, too, there is an excess of muco-pus, and the urine tends to become alkaline.

In hæmorrhage from the *prostate or urethra* the blood is florid, escapes often in the intervals between micturition, or appears as a small clot at the commencement of the act.

In addition to the above-named local diseases, hæmaturia may attend purpura, scurvy, the continued fevers, or other general diseases; it may be caused by the presence of the Bilharzia hæmatobia, or by the action of some poison on the kidneys, like cantharides or turpentine. After repeated doses of sulphonal the urine may acquire a Burgundy-red colour, due to the presence of hæmatoporphyrin, a derivative of hæmoglobin.

Hæmoglobinuria.—The urine contains hæmoglobin, but no blood corpuscles. It is dark red when viewed in bulk, or it resembles strong tea or porter, and is generally turbid, yielding a chocolate-coloured or black sediment. It is always albuminous, and when boiled the albuminous substance forms a small brownish clot, which tends to float upon the surface (thus differing from serum albumin, which is precipitated in flakes, which coalesce to form a thick white precipitate). In the sediment, granular matter, minute crystals of hæmatine and oxalate of lime, granular and fibrinous tube casts, and very rarely a few blood corpuscles are found. The possibility of other blood corpuscles having been dissolved or disintegrated into granular matter must always be borne in mind.

Hæmoglobinuria occurs sometimes in connection with fevers, as enteric or scarlet fever; sometimes it is the result of certain poisons, especially chlorate of potash; rarely it has been found in new-born infants in association with cyanosis and icterus. Occasionally it occurs intermittently, constituting the peculiar affection known as **paroxysmal hæmoglobinuria**. This is chiefly met with in adult males, and most commonly after exposure to cold. The attack begins with a slight rigor, then urine, charged with the colouring matter of blood, is passed. The symptoms, however, quickly subside, and in a few hours the urine becomes perfectly natural, and the patient often remains in good health till the next seizure. Towards the end of an attack a sub-icteric tint of skin may be noticed; during the early chilly stage, a condition of "local asphyxia," constituting Raynaud's disease, is not uncommon—the fingers, toes, nose and ears become cold, white, or livid, and sometimes even gangrenous.

Bile.—Of the elements of bile, two are met with in the urine—bile pigments and the salts of the bile acids. A third element, cholesterin, has not been detected in jaundiced urine.

Bile Pigments always tinge the urine, the colour varying from a yellowish-brown to a brownish-red, or it may even be as dark as porter; there may be also a greenish shade, best seen on the surface. The froth produced by shaking is more permanent than with healthy urine, and it is yellow or greenish in colour. White filtering paper and linen are stained yellow by the urine.

Tests.—1. **Gmelin's.**—(*a.*) On to the surface of a small quantity of fuming nitric acid (containing nitrous acid) in a test-tube, a little of the urine is allowed to trickle by means of a pipette. A green ring is seen at the contact zone if biliary colouring matters are present, and below the green, blue, violet, red and yellow rings may be seen in the order named from above downwards. The green colorisation is the most constant, and is the only certain sign of the presence of bile; the other colours may be developed in the absence of jaundice, as when indican is present. If the biliary pigments have undergone change from decomposition of the urine, the test may fail.

(*b.*) A similar play of colours is also obtained by allowing a drop of the urine and a drop of the nitric acid to run together on a white plate, but the green colour is less perfectly seen than in (*a*).

(*c.*) Dip a piece of white filter-paper into the urine, and let a drop of fuming nitric acid fall on the moistened part; at the edge of the yellow bile-stain an intense green is seen, which passes into blue, and, still farther away, into a faint pink colour.

2. **Marechalt's Test.**—Drop some tincture of iodine carefully on to the urine in a test-tube; a green colour appears where they touch. If they are now mixed, the whole becomes of a fine green colour. Concentrated urine should be first diluted with water, and an excess of iodine must be avoided.

Note.—1. Bile pigment is often present in the urine before the skin is perceptibly discoloured; on the other hand, the skin and mucous membranes often remain icteric long after the urine has become free from bile.

2. An examination of bilious urine does not throw light on the cause of the jaundice; commonly there is obstruction at some part of the biliary passages, but the functions of the bile secretion may be perfectly normal, and yet from change in the colouring matter of the blood bile pigment may be present in the urine.

3. It is also noteworthy that in most bilious urines a few casts of the renal tubules will be found; they are chiefly epithelial and hyaline, usually of small size, and are stained yellow.

4. Acidification of a bile-stained urine, either with a drop of nitric acid or of acetic acid, often produces a slight cloud, which is due in some cases to the precipitation of mucin, in others to an albuminous

body. In testing the urine by the contact method with cold nitric acid, there is often a white cloud just above the line of contact, higher than the ordinary precipitate in albuminous urine.

Bile Acids are present in the urine of jaundice; in larger quantity in recent than in old cases, but their clinical importance has not yet been demonstrated.

Test.—To a test-tube half full of strong sulphuric acid add a few grains of grape-sugar, and float a little urine on to the surface of the mixture; a purple or violet colour at the line of junction indicates the presence of bile acid.

Glycosuria.—Minute traces of sugar are said to exist in healthy urine, but not in sufficient amount to respond to the ordinary tests. When sugar can be detected in the urine by the tests described below, it is well to regard it as a pathological sign of grave significance, and all possible causes and associated symptoms should be carefully investigated.

Cases of glycosuria may be separated into two groups:—

1. **Incidental, Temporary, or Intermittent Glycosuria.**—In many elderly persons distinct evidence of sugar may be found in the urine passed shortly after breakfast. Also during the convalescence from an acute disease, such as cholera, or after a paroxysm of ague, epilepsy, hysteria, whooping-cough, or asthma. Grape-sugar has also been found during an attack of sciatica; after injury to the brain; in cerebro-spinal meningitis; in cirrhosis of the liver, and also occasionally after poisoning with morphia or chloroform.

2. **Persistent Glycosuria.**—The habitual presence of a large quantity of sugar in the urine is the essential feature of Diabetes mellitus.

Tests.—1. **Moore's or Heller's Test.**—Add an equal volume of liquor potassæ or liquor sodæ to the urine in a test-tube and boil the upper layer of the mixture; the heated portion gradually darkens in colour, varying from yellow to black, according to the quantity of sugar present or the duration of boiling. For this test to yield positive results, there should be at least 0.3 per cent. of sugar or 1½ grs. to the ounce.

Precautions.—(1.) If much albumin is present, boil and filter.

(2.) All high-coloured urines deepen slightly in colour when boiled with liq. potassæ.

2. **Trommer's Test** depends upon the fact that in alkaline solutions sugar reduces a metallic oxide to a suboxide. To a little urine in a test-tube add nearly an equal bulk of liq. potassæ or liq. sodæ and a few drops of a weak solution of cupric sulphate: a bluish precipitate of hydrated cupric oxide appears, which redissolves on shaking, giving a clear blue liquid, but if sugar is absent a turbid green. On boiling

the mixture, if sugar is present, a yellow and finally a red precipitate of cuprous oxide is produced. A reliable result is obtained in the presence of from 0.5–1 per cent. of sugar.

Precautions.—(1.) Remove any albumin present.

(2.) Avoid adding too much cupric sulphate.

(3.) Avoid prolonged boiling. Reduction of cupric oxide may be caused by other substances besides sugar, especially by glycuronic acid, by excess of uric acid, or of mucus; it may be hindered, when the urine contains only a small quantity of sugar, by the presence of ammonium chloride, or of other ammonia salts.

3. **Fehling's Solution.**—Here the copper and the alkali are mixed in the exact proportion necessary. The solution is prepared from the following prescription :—

Crystals of cupric sulphate, 34.64 grms. ; neutral tartrate of potash, 173 grms.; solution of soda (sp. gr. 1.12), 480 c.c. Add water to make up 1000 c.c.

Every 10 c.c. corresponds to 0.05 grm. of grape-sugar.

Boil a little of this solution in a test-tube, then, if no deposit occurs, the solution is in good condition. Now add a drop or two of the suspected urine. If it contain much sugar, the suboxide of copper falls as a brick-red precipitate at once; if several drops of urine be added, the precipitate is yellow. If no precipitate occurs, add more urine until a bulk equal to, *but not greater than*, that of the Fehling's solution has been poured in, and boil again; then if a small quantity of sugar be present, the solution turns a yellowish-green, and a bright yellow deposit slowly subsides.

If, however, the urine contain less than half per cent. of sugar, the precipitate only appears as the liquid cools, when the mixture gradually assumes a green, milky appearance.

Precautions.—(1.) Remove albumin as in Trommer's test.

(2.) The test solution should always be boiled first, for it is apt to decompose by keeping, some of the tartaric passing into racemic acid, which has a reducing action on the oxide of copper.

(3.) The quantity of urine should never exceed that of the test solution.

(4.) Prolonged boiling should be avoided, for any urine, especially if high coloured, when boiled some time with an excess of the copper solution, will turn a greenish-yellow colour, due to the reduction of copper by some other organic substance.

Since the reduction of Fehling's solution on boiling it with urine may be due to other substances than sugar, this only affords presumptive proof of the presence of sugar; the fermentation test alone affords positive proof.

Quantitative Estimation.—(*a.*) Into a burette introduce 10 c.c. of the urine, and dilute with water up to 100 c.c.

(*b.*) Dilute 10 c.c. of Fehling's solution (representing 0.05 grm. of sugar) with about twice its volume of water, and place it in a flask on a wire-gauze support under the burette.

(*c.*) Boil the diluted Fehling's solution, and whilst it is boiling drop in the diluted urine till all the cuprous oxide is precipitated as a reddish powder and the blue colour has entirely disappeared, the supernatant fluid becoming straw-yellow in colour. This is determined by allowing the precipitate to subside and then holding the flask between the eye and the light.

(*d.*) Read off the number of c.c. of diluted urine which were dropped into the flask. If 36 c.c. were used, they represent 3.6. c.c. of the undiluted urine, which therefore contains 0.05 grm. of sugar. The number of grammes contained in 100 c.c. of urine, or in the number of c.c. passed in the twenty-four hours, can then be easily calculated.

Thus, if 1600 c.c. of urine were passed :

$$\text{then } 3.6 : 0.05 :: 1600 : x$$

$$\frac{1600 \times 0.05}{3.6} = \frac{20.0}{.9} = 22.22 \text{ grammes}$$

$$\text{or per cent.} = \frac{0.05 \times 100}{3.6} = 1.4$$

4. **Pavy's Solution** is composed as follows :—

Cupric sulphate	4.158 grammes
Potassic sodic tartrate	20.4 ,,
Potash (caustic)	20.4 ,,
Strong ammonia (sp. gr. 0.800)	300 cb.c.
Water to 1 litre	

The tartrate of potash and the caustic potash are dissolved in a portion of the water, and the sulphate of copper with the aid of heat in another portion. The latter solution is then poured into that of the potash salts, and when cold the ammonia is added ; finally, water to the required amount. Of this solution 10 c.c. are reduced by 0.05 grms. of glucose. The reagent is used precisely in the same manner as Fehling's solution. Its *advantages* are : (1) that the action of sugar is shown by the discharge of the blue colour of the solution, not by precipitation of the cuprous oxide, which is held in solution by the ammonia ; hence the end-point of the reaction is more easily determined than with Fehling. (2) Pavy's solution is more stable than Fehling's solution.

Precautions.—Avoid dissipation of the ammonia by passing the point of the burette through a cork fitting the mouth of the flask, the cork

having another opening for escape of steam. When the blue colour has entirely disappeared and the solution is as colourless as water, the reduction is complete. On cooling, the solution again acquires a blue tint, the cuprous oxide held in solution by the ammonia being rapidly reconverted into cupric oxide. The test may be purchased in the form of pellets or enclosed in glass tubes, each containing 10 c.c.

5. **Fermentation Test.**—Occasionally in testing for sugar with Fehling, a doubtful result is obtained; the colour may change to a purplish-green, or there is an imperfect reduction after long boiling. In such cases the fermentation test is of great value, for if on adding a few crumbs of German yeast to a test-tube full of urine, and inverting over a saucer placed in a warm place, there is an evolution of carbonic acid gas, sugar is undoubtedly present; if the reaction is negative, sugar is absent.

Precaution.—Perform a blank experiment with yeast and water at the same time, for some specimens of yeast evolve bubbles of gas; if this occurs, the volume of gas thus yielded may be compared with that resulting from the yeast and urine.

Quantitative Estimation.—In ordinary clinical work Sir William Roberts's differential density method is by far the easiest, and is practically accurate.

A piece of German yeast the size of a small walnut is added to about 4 oz. of the saccharine urine in a 12-oz. bottle, which is closed with a cork in which a nick is cut so as to allow the carbonic acid to escape. The bottle is placed on the mantelpiece or other warm place, and beside it is placed a tightly corked 4-oz. bottle filled with some of the same urine, but without yeast. After eighteen to twenty-four hours the specimens are respectively poured into two urine glasses, and when cool the sp. gr. of each is taken. The number of degrees difference in density between the fermented and the unfermented urine indicates the number of grains of sugar per fluid ounce. For example, if the sp. gr. of the original urine is 1050 and that of the fermented 1005, the number of grains of sugar in a fluid ounce will be 45. If this be multiplied by 0.23, the percentage quantity 10.35 is obtained. The urinometer should have a long scale, in order that the sp. gr. may be read with great accuracy.

6. **Picric Acid Test.**—To a little of the suspected urine in a test-tube, add nearly an equal bulk of a saturated solution of picric acid and a small quantity of liquor potassæ. Boil, and if sugar is present the mixture turns a dark mahogany colour. Normal urines, especially if concentrated, also turn a darker hue, but this is much less marked. If the urine contain albumin or globulin, a precipitate is formed, but this does not interfere with the reaction for sugar.

The exact depth of colour, estimated by comparison with a standard coloured solution, is used by Dr. George Johnson for quantitative estimations.

7. **Phenyl-hydrazin Test.**—Fill a test-tube three parts with the suspected urine, and add about 7 or 8 grains of phenyl-hydrazin hydrochloride and about twice as much sodium acetate. Place the tube in a small beaker filled with water, which is kept boiling for from half an hour to an hour. On cooling, if sugar is present, crystals of phenyl-glucosazone fall to the bottom of the tube, from which they can be removed by a pipette, placed on a glass slide with cover-glass, and examined under the microscope. The crystals are needle-shaped, and are usually agglomerated so as to form small spherules; some may be seen isolated or in small groups (see Fig. 185).

FIG. 185.—Phenyl-Glucosazon Crystals from Diabetic Urine. (*v. Jaksch.*)

Acetonuria.—Acetone is found in the urine of patients suffering from severe or advanced diabetes, but its presence does not always forebode the onset of coma, as was formerly supposed. Acetonuria also occurs in connection with febrile diseases, Bright's disease, cerebral disorders, cancer, and sometimes in simple inanition; indeed, traces of acetone can be detected in healthy urine.

Tests.—(1.) Add a few drops of a freshly-made strong solution of nitro-prusside of soda to the suspected urine, also a few drops of liquor potassæ. If acetone is present a ruby-red colour is produced, which slowly fades to a straw-colour, but gives place to a violet or purple hue on acidulation. (2.) Add to the urine a few drops of an aqueous solution of iodine (dissolved with the aid of potassium iodide), and a little caustic potash. On gently warming, a precipitate of iodoform crystals takes place. It is to be remembered that ethyl-alcohol yields a like reaction.

other diseases gives an identical colour with the sulphanilic acid solution; moreover, the reaction is not constant in cases of typhoid, and hence cannot be regarded as of much diagnostic value.

Urea.—The principal final product of the metabolism of nitrogenous compounds is urea, in which form nearly 90 per cent. of the total nitrogen present in the urine is excreted. The daily amount varies with the quantity of proteids assimilated; in an adult male it ranges from 30 to 40 grammes, the average being about 33 grammes (500 grains). Urea is present in normal urine from an adult at the rate of about 2 to 3 per cent. In proportion to the body weight, children excrete more than adults, the absolute quantity being less in women and children than in men. Children excrete about 1 gramme per kilo. of body weight, and adults 0.4 to 0.6 gramme. The excretion of urea is increased in three or four hours after the intake of food containing much proteids. It is increased by greater activity of metabolism, *e.g.*, in the stage of fever, or of acute inflammation which precedes the climax, after which it is diminished. Increased formation of urea does not necessarily imply increased excretion; it may be retained in part, to be excreted subsequently. Copious draughts of water—especially under the conditions last named—increase the excretion of urea. Conversely, diminished metabolism lessens the output of urea; hence in most chronic diseases which are attended by imperfect nutrition the amount of urea is below the normal. In acute atrophy of the liver, and in acute phosphorus poisoning, the excretion of urea is lessened although the total amount of nitrogen may not be much, if at all, below the normal, the difference being made up in acute atrophy by intermediate products, as leucin and tyrosin, and in phosphorus poisoning chiefly by ammonia. As previously stated, the amount of urea formed may be increased, but it may be only partially excreted; therefore the amount present in the urine may be less than normal although metabolism is going on as usual, as is the case in uræmia.

When nitric acid is added to urine rich in urea, a crystalline deposit of urea nitrate forms, as it is much less soluble in water than urea. To ascertain the amount of urea present, it is most convenient, for clinical purposes, to decompose it and to measure the nitrogen set free. Any form of nitrometer may be used for this purpose, but for simplicity and convenience the instrument devised by Doremus excels (Fig. 186). It consists of a graduated tube, closed at the upper end and curved at the lower end, which is furnished with a bulb. A specially formed 1 c.c. pipette is supplied with the apparatus. The reagent consists of a solution of hypobromite of soda containing soda in excess. It is prepared by dissolving 100 grammes of sodium hydrate

in 250 c.c. of water. To 23 c.c. of this solution 2 c.c. of bromine are added, and the mixture is gently agitated until the bromine is dissolved. The solution, when cold, is poured into the ureometer so as to fill the tube to the mark on the bend below the bulb, and sufficient water is added to fill the lower portion of the bulb. The pipette is charged with the urine to be examined up to the mark—1 c.c.; the pipette is introduced into the ureometer as far as the bend, and the urine expelled by compression of the rubber nipple. The urea is decomposed into carbon dioxide, nitrogen and water. The carbon dioxide combines with the soda, and the nitrogen collects at the upper part of the tube, which is graduated, so that the amount of urea present in the cubic centimetre of urine can be read off. Each division indicates 0.001 gramme of urea. The percentage is obtained by multiplying the result of the experiment by 100. The total quantity of urea excreted in twenty-four hours is found by multiplying the result of the experiment by the number of cubic centimetres of urine which were passed in the twenty-four hours. If the urine contains albumin, it must be separated by boiling before estimating the urea.

FIG. 136.—Ureometer of Doremus.

The solution of hypobromite should be freshly prepared, as it decomposes into bromate on keeping.

If preferred, the B.P. liquor sodæ chlorinatæ may be substituted for the hypobromite solution.

When greater accuracy is desired, an apparatus should be used that permits of exact adjustment of the water level; correction also must be made for temperature, barometric pressure, and the tension of aqueous vapour.

The amount of urea in urine may be estimated by volumetric analysis, but for clinical purposes the method is not so convenient, nor is it so accurate as the one described.

SEDIMENTS AND MICROSCOPICAL EXAMINATION OF THE URINE.

UNORGANISED SEDIMENTS.

Uric Acid does not exist in the free state in normal urine, but under certain conditions it may be deposited as orange-red crystals. These crystals are usually large enough to be seen by the naked eye; they look

like grains of cayenne pepper as they lie at the bottom or adhere to the sides of the urine glass. Rarely, they are too small to be seen without the microscope, and occasionally are almost colourless.

Recognition.—Under the microscope the crystals are seen to vary in form. Typically lozenge or whetstone-shaped, they may appear as hexagonal plates, or present a rough resemblance to "dumb-bells;" sometimes single, they are often aggregated, forming stars, fan-shaped figures, or rosettes (Fig. 187).

Tests.—1. The crystals are dissolved by liquor potassæ or other alkali, and reprecipitated in hexagonal plates by excess of hydrochloric or acetic acid.

FIG. 187.—Forms of Uric Acid. 1. Rhombic plates; 2. Whetstone forms; 3. Quadrate forms; 4, 5, Prolonged into points: 6, 8. Rosettes; 7. Pointed bundles; 9. Barrel forms precipitated by adding hydrochloric acid to the urine. (*Landois and Stirling.*)

2. They are the only urinary crystals that invariably show colour under the microscope.

3. The **murexide test.**—Place some crystals of uric acid in a porcelain dish, add a drop or two of strong nitric acid, and dissolve the crystals by gentle heat, which is maintained until the deposit is dry. When cool, touch the yellowish-red residue with a drop of dilute liquor ammoniæ, when a purple-red appears, which, on the addition of a drop of liquor potassæ, becomes more blue.

4. A uric acid sediment is not dissolved on heating the urine—a distinction from urates.

5. It is not dissolved by acetic acid—a distinction from phosphates.

6. When boiled for some time with cupric sulphate and liquor potassæ, uric acid partially reduces the copper salt to cuprous

oxide. This, as a fallacy in testing for sugar, has been already mentioned.

Clinical Import.—Healthy urine, after long standing, deposits uric acid; if the deposition occurs *immediately* after the urine is passed, it suggests the probable formation of calculi in some part of the urinary passages.

The daily excretion of uric acid is much increased in leukæmia, after an attack of gout, in certain diseases of the liver, in scurvy, &c. It is diminished during the paroxysm of gout.

Amorphous Urates.—A loose, reddish deposit, consisting of amorphous urates (*i.e.*, uric acid in combination with potassium, ammonium, sodium, and usually lime), is the most common of all urinary sediments. Its colour, due to the affinity of amorphous urates for the colouring matter of the urine, varies from fawn to pink, according to the density of the urine; deep-coloured urines of high specific gravity deposit a red brick-dust-like sediment. In children urates are often white, producing the "white-like-milk" urine. A purplish film or bloom is frequently seen on the surface and sides of the urine, and is highly characteristic of amorphous urates. Urine containing an excess of amorphous urates is clear when passed; the muddiness appears on cooling.

Micro-chemical Characters.—(1.) Under the microscope the deposit is seen to consist of minute granules, sometimes closely aggregated; if adherent to mucous shreds, some resemblance to granular casts is produced, but the variable breadth and the irregular and indistinct contour of such agglomerations serve to distinguish them from true casts (see Fig. 194).

(2.) The deposit at once disappears on warming the urine. By this test the white urates of childhood are easily distinguished from phosphates.

(3.) Crystals of uric acid may not only be obtained from urates by the addition of an acid, but also by simply treating the deposit with warm water, for uric acid is present much in excess of the amount necessary to combine with the bases present, and so the loosely combined excess is easily separated.

(4.) The sediment reacts to the murexide test.

Clinical Import.—A deposit of urates is met with in a large number of organic and functional diseases, especially when there is pyrexia, *e.g.*, in pneumonia and in rheumatic fever. In health anything (such as sweating, fasting, &c.) that concentrates the urine tends to cause a deposit. The urine is often heavily loaded with urates in cirrhosis of the liver.

Crystallised Urates.—(1.) **Urate of Soda.**—The somewhat rare

spontaneous deposit of urate of soda is seen under the microscope to consist of irregular spheres, either simple or furnished with spiny projections. It is frequently deposited from the urine contained in the bladder, and is thus liable to cause irritation of the mucous membrane

FIG. 188.—Urate of Sodium.

of the bladder or of the urethra. The **acicular** crystals met with in gouty concretions are never deposited spontaneously in the urine.

(2.) **Urate of Ammonia,** the only urate found in alkaline urine, is sometimes precipitated along with phosphates in ammoniacal urine. It is generally whitish in colour. Under the microscope globular masses or "thorn-apple" spherules, or sometimes minute slender dumb-

FIG. 189.—Urate of Ammonium, Sediment in Alkaline Fermentation. (*v. Jaksch.*)

bells, are seen. Being opaque, they appear dark coloured by transmitted light.

An **Oxalate of Lime** deposit, usually very scanty, can be recognised by the naked eye as a fine powder or a silvery line on the top of a delicate cloud of mucus—the "powdered-wig" looking sediment. If a specimen of urine which yields a deposit of oxalate of lime is allowed

to stand in a urine glass, white lines appear on the sides of the glass, as though it were scratched; the lines are due to linear deposition of crystals. Most frequently met with in acid urines, the crystals are sometimes seen in neutral or faintly alkaline urine, and occasionally alongside crystals of the triple phosphate.

Recognition.—Oxalate of lime crystals occur in two forms:—(1.) The commoner are octahedra, looking like a square-folded envelope; more rarely they appear as quadrilateral columns with pyramidal ends. (2.) Occasionally dumb-bell shapes are seen, which much more closely resemble real dumb-bells than the "dumb-bell" shaped crystals of uric acid. Oxalate of lime crystals often show a tendency to form microscopic concretions.

Tests.—(1.) Oxalate of lime is insoluble in acetic acid and in alkalies, but is soluble in hydrochloric acid. (2.) The form of the crystals is characteristic, so that only exceptionally can they be mistaken for other crystals. Sometimes very small crystals of the triple phosphate resemble the octahedra of oxalate of lime; a drop of acetic acid quickly

FIG. 190.—Oxalate of Lime from Sediment in a Case of Cystitis and Pyelonephritis. (v. Jaksch.)

dissolves the former, but leaves the latter unchanged. Sometimes, when of large size and bile-stained, they present a superficial likeness to uric acid crystals.

Clinical Import.—Deposits of oxalate of lime are often found in the urine of healthy persons, especially after a meal containing rhubarb, tomatoes, &c. When constant and large, they point to impaired digestion, to debility or a depressed condition of the nervous system. The crystals are also met with in catarrhal jaundice, in diabetes mellitus, in convalescence from some diseases, especially typhus, and in paroxysmal hæmoglobinuria.

Earthy Phosphates form the ordinary bulky white sediment of ammoniacal urine, being often mixed with mucus and pus. Urine which deposits phosphates is not always alkaline, it may be neutral or even feebly acid. On gently heating in a test-tube the urine becomes turbid and clears again on cooling, but if boiled a chemical change occurs, and the turbidity does not vanish on cooling. A drop of acetic acid at once dissipates the turbidity, showing the absence of albumin.

The earthy phosphates are the triple phosphates and the phosphate of lime.

1. The Triple or the Ammonio-Magnesian phosphate forms a white sparkling crystalline deposit; the crystals stud the sides of the urine glass and make an iridescent scum on the surface. The ordinary

FIG. 191.—Triple Phosphate Crystals from Sediment in a case of Chlorosis. (*v. Jaksch.*)

form is a triangular prism with bevelled ends, presenting, however, many modifications; they are the largest of urinary crystals. Star-shaped feathery crystals are also sometimes seen.

2. Phosphate of Lime occurs in two forms: (*a.*) An **amorphous** white flocculent deposit, showing under the microscope small pale

FIG. 192.—Stellar Phosphates.

granules associated with crystals of the triple phosphate. The urine, always alkaline, often has an iridescent film on its surface. The amorphous phosphate may be present in the alkaline urine passed shortly after a meal.

(*b.*) **Crystalline Phosphate of Lime or Stellar Phosphate.**—The

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

ORGANIC DEPOSITS.

Blood Corpuscles.—Their appearance and the duration of their visibility varies with the density and the reaction of the urine. In an acid urine of normal specific gravity, the red discs are easily recognised by their characteristic biconcave shape, and by their colour—which, however, is paler than in pure blood; they may remain unaltered for several days. In a urine of low density they lose their central depression and become spherical; and when the specific gravity is very low, or if the urine be ammoniacal, they often disappear very speedily. In highly concentrated urine the concavity is marked, the corpuscle shrinks and assumes a crenate or horse-chestnut shape. Sometimes (especially, it is said, when the bleeding is from the kidneys) the corpuscles exhibit amœboid movements—throwing out and drawing in processes. They are only exceptionally, as when there is a considerable hæmorrhage from the bladder, seen in rouleaux; as a rule they are discrete. They are distinguished from other bodies by their feeble refractive power, their fine delicate outline, and by the absence of a nucleus.

Mucous and Pus Corpuscles are spherical cells slightly larger than a red blood disc; they closely resemble white blood corpuscles. The **Mucous** corpuscle, usually ill-defined and granular, often contains a simple nucleus: every grade is seen between it and an epithelial cell.

The **Pus** corpuscle exhibits a multiple, usually a tripartite, nucleus after the addition of a drop of acetic acid.

Both mucous and pus corpuscles are rapidly dissolved by caustic alkalies; hence if a portion of the glairy mass found in ammoniacal urine be put under the microscope, no pus cells are visible, or only a few black dots, the remnants of their nuclei.

To distinguish pus from round epithelial cells, add a few drops of a solution of iodine in iodide of potassium, when they turn a mahogany brown; epithelial cells turn a pale yellow colour.

Epithelium.—From the healthy genito-urinary passages of the male but few epithelial cells can be detected in the urine, but in the female squamous cells from the vagina are almost constantly present. In disease, epithelium from all parts of the passages may be seen. The slight differences which normally exist between the cells from different parts of the renal tubules, or between those of the pelvis, ureter and bladder, can rarely be made out after the cells have soaked in the urine; the source is determined by the prevailing type of cell, by the presence or absence of casts, and by the accompanying symptoms. It is impossible to say whether a particular renal cell comes from a

convoluted or from a collecting tubule; this, however, is of little practical importance, for it is rare for an inflammatory process to be limited to such a restricted portion of kidney. The following three varieties of epithelium may be easily distinguished.

1. **Round Cells** with a well-defined single nucleus may come from (*a*) the urinary tubules, especially their convoluted portions; or (*b*) from the lower layers of the mucous membrane of the pelvis, ureter and bladder. They are larger than leucocytes, and the nucleus is usually visible without reagents, and is thus distinguished from the multiple nucleus of a pus cell, which requires acetic acid before it appears. In diseased conditions the renal epithelial cells may be wasted or broken up into amorphous granular matter, or they may contain oil globules, &c.

2. **Columnar Epithelium.**—Cylindrical, tailed and spindle cells, with well-defined nuclei, are mainly derived from the pelvis, ureter, or urethra. Epithelium from the pelvis of the kidney is never found in

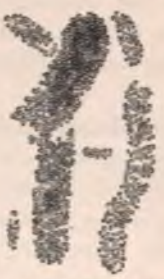


FIG. 194.—Casts of Urates, from a Case of Emphysema. (*v. Jaksch.*)

healthy urine; its presence always indicates irritation of that part. Many of the pelvic cells closely resemble cancer cells, but the latter are usually more numerous, larger, and more perfect than the former. The diagnosis, however, between cancer and calculous disease of the kidney is more reliably based on other symptoms.

3. **Squamous Epithelium** comes from the bladder or vagina; it consists of large irregular-shaped cells containing a simple nucleus. Those from the vagina are larger and more apt to occur in flakes than the cells from the bladder.

Casts.—Inorganic Casts, or cylindrical forms composed of an aggregation of amorphous urates, amorphous phosphates, or of hæmatoidin crystals, &c., are occasionally met with in adults as well as in children, but especially in the urine of new-born babes. Organic casts form by far the more important group; these are moulds of the tubules, for the most part composed of an albuminous material which has escaped from the capillaries, and solidifies in the tubules, entangling any loose

cells or other elements that may be present; it then contracts, passes along the tubule to the pelvis, and thus escapes with the urine. The size of the casts, and the presence or absence in them of various elements, are points by which they are classified.

FIG. 195.—Epithelial Cast.

1. **Epithelial Casts.**—Sometimes, as in scarlet fever, the renal cells are simply heaped together in the form of a cylinder; but more commonly the cast is composed of coagulable material containing epithelial cells.

2. **Blood Casts.**—Here also red or white corpuscles may amalgamate to form a cast, but more frequently they are found irregularly studding a fibrinous mould. When composed of white blood corpuscles they resemble pus casts; the latter are occasionally seen in abscess of the kidney.

The presence of the above cell-formed casts (1 and 2) in the urine points to acute nephritis or to an acute exacerbation of a long-existing nephritis.

3. **Granular Casts** arise mostly from the destruction of epithelial and blood cells. Their colour varies from a yellowish-white to a brownish-red; their size and contour also vary, and they often occur in broken pieces with sharp edges; they are studded with granules, sometimes very fine, sometimes very coarse. When there are

FIG. 196.—Blood Cast.

FIG. 197.—Granular Cast with a few Fat Drops on Surface.

but few and very fine granules the transparent hyaline mould is visible, but when the granules are numerous the cast looks dark and opaque. The dark coarsely granular casts are generally about $\frac{1}{100}$ inch in diameter. Sometimes blood discs or fat drops are seen on their surface.

These casts are significant of an inflammatory process going on in the kidney. They are, however, found, though very rarely, in the urine of cyanotic induration of the kidney.

4. **Hyaline Casts** are glassy-looking, often with ill-defined contour, and are sometimes too transparent to be seen without the addition of a coloured fluid, such as a watery solution of iodine, or of magenta, or aniline violet, &c. They vary much in length; they are from $\frac{1}{3000}$ to $\frac{1}{500}$ of an inch in width. Sometimes they show a tendency to branch at their ends; occasionally their contour, instead of being straight, is wavy. Sometimes homogeneous throughout and free from deposits, they more commonly have—(1) a few epithelial cells, normal, containing oil drops, or converted into granules, on their surface; or (2) a few red blood discs or leucocytes; or (3) possibly a few crystals, as of oxalate of lime, are seen studding their surface.

FIG. 198.—Fatty Casts.

Darker looking hyaline casts are sometimes called "waxy;" they are solid-looking and highly refractive; some are very long, others are represented by short and broad fragments, which are often cleft and broken. They may be quite homogeneous or covered with various elements. Occasionally they react to the albuminoid test, but often do not even when the kidneys are lardaceous; they may give the albuminoid reaction through changes produced by a long duration in the urinary passages. The above casts, hyaline and waxy, are found in every variety of nephritis.

5. **Fatty Casts** are transparent or dark granular casts, which are dotted over with minute oil drops. Sometimes the oil particles are collected into dark cylindrical masses, and from their surface radiating needles (composed of salts of the higher fatty acids) are occasionally seen to project.

6. **"Cylindroids"** are long, ribbon-shaped forms, of variable breadth and contour, which have been found in the urine of scarlet fever, also sometimes in other cases of nephritis. They are probably mucoid in nature, and are of no diagnostic importance.

The Clinical Significance of Casts.—Normal urine is free from casts, though very exceptionally a few small hyaline ones may be found; the presence of many hyaline casts points to a severe disturbance of the general circulation or to some kidney irritation. It is difficult to form an accurate estimation of the condition of the kidneys from a study

merely of the casts passed in the urine; still a knowledge of the prevailing type—whether granular or fatty, for example—is of considerable assistance in making a diagnosis. Specimens of urine passed on different days must be repeatedly examined before a correct judgment can be formed as to the prevailing types. Epithelial and blood casts with a plentiful desquamation of renal epithelium point to an acute inflammation of kidney structure; oil drops in the epithelium or scattered over hyaline casts indicate that a fatty change is going on in the kidney. Hyaline casts are met with in both recent and old cases of nephritis; large hyaline casts suggest that the renal tubules have become widened through loss of their epithelium; they are found in association with granular casts in chronic cases, also in the terminal period of acute scarlatinal nephritis. Tube casts are abundant in acute parenchymatous nephritis, less abundant in chronic parenchymatous nephritis, and are usually scanty in lardaceous disease; in congestion of the kidney also there are very few. In obscure cases of bloody or purulent urine the presence of casts suggests a renal element in the causation of the blood or pus.

In the large majority of cases when casts are found, the urine contains albumin; exceptionally, it cannot be detected by the most delicate tests. Thus, sometimes in passive renal congestion from mitral disease, small hyaline casts, with or without granules, are found when albumin is absent. In chronic Bright's disease albumin may temporarily disappear, and yet casts may be found in the deposit, just as in the convalescence from acute Bright's disease after the disappearance of albumin. In icterus, too, as already stated (see p. 324), casts are frequently present; a trace of albumin is often also present.

Diagnosis.—As a rule, casts are easily recognised; occasionally one of the following forms may be mistaken for a cast.

(1.) Mucous coagula studded with urates may resemble granular or fatty hyaline casts (see Fig. 194).

(2.) In spermatorrhœa, &c., hyaline cylindrical forms (which enter the prostatic part of the urethra from the vasa deferentia and seminal tubes) may be present. They are distinguished by their greater size (being twenty or thirty times wider than kidney casts), and by the absence of albuminuria, and of other renal symptoms.

(3.) Cylindrical collections of micrococci, found when a septic pyelitis affects the kidney substance, may resemble granular casts, but the micrococci resist reagents, such as liq. potassæ or nitric acid. The dotting also is very fine and evenly distributed; it is much more regular than in the granular cast.

(4.) In new-born children, and occasionally in adults, cylindrical masses of urate of ammonia may be seen in the urine. They are soon

dissolved by a drop of acetic or hydrochloric acid, and are replaced by crystals of uric acid.

Search for Casts.—The specimen of urine should be fresh, for if twenty-four hours old, all the casts may have been dissolved.

When it has stood for a few hours in a conical vessel, the sediment may be examined. In order to exclude the action of air in the sediment, the conical glasses in which the urine is put to settle should have ground-glass covers to fit their own ground upper edges.

The urine should be decanted, and the deposit dropped on to a slide, or, without decanting, a little of the deposit may be withdrawn by a pipette; it is very important that the pipette should be quite clean.

Sometimes, owing to the density of the urine, casts are deposited slowly; in such a case dilution of the urine will facilitate their descent.

If no casts, or only a few, are seen, it will be well to obtain a specimen of the deposit from the whole twenty-four hours urine; or, if the urine tends to become alkaline (in which case casts are soon dissolved), successive portions of the urine as passed are allowed to stand a short time and then examined. In this way in doubtful cases the whole of the twenty-four hours deposit may be investigated without running the risk of solution of the casts through long standing.

Casts may be present when there is little or even no deposit appreciable to the naked eye, and rarely in the absence of albumen. In rare cases albumen may be present in large quantity, and yet no casts can be found.

CHAPTER XI.

EXAMINATION OF PUNCTURE FLUIDS.

In diseased conditions, fluids may accumulate in the various cavities of *the body*, in the subcutaneous tissues, or in cystic new formations. *Again, collections of purulent fluid* may be formed in various organs *and cavities.*

The presence and character of a collection of fluid may be determined by *puncturing the affected region* with the needle of a hypodermic syringe, *and, if fluid is present, drawing off* a small quantity of it for examination. *If strict antiseptic* precautions have been used, no bad results *will follow, even should no fluid* be present.

The collection of fluid may be the result of inflammation, in which case it is termed an exudation; or it may be due to abnormal or to obstructed circulation in the parts affected; the fluid would then be termed a transudation. Although the character of the two classes of fluid differs, it is sometimes very difficult to say to which class a given specimen belongs.

EXUDATIONS.

Exudations may be serous, hæmorrhagic, putrid, sero-purulent, or purulent.

Serous Exudations are yellowish, and more or less turbid, the turbidity being due to the presence of cell elements; when allowed to stand, a clot forms which contains much fibrin. The clotting occurs sometimes directly the fluid is withdrawn, sometimes a little later, but, in any case, within twenty-four hours. Such fluids contain a few scattered red blood corpuscles, leucocytes and endothelium cells; their reaction is alkaline, and their specific gravity is generally above 1018. They are rich in serum albumin and serum globulin; small quantities of uric acid and of sugar are present. Serous exudations are met with in the pleural cavity after pleurisy, in the pericardium after pericarditis, in the abdominal cavity after peritonitis.

In appearance serous exudations strongly resemble transudations, but they differ in the following points:—In serous exudations the fluid is more turbid and the cell elements are more numerous; there is a greater tendency to coagulate; the specific gravity is higher, and the amount of albumin greater.

Exudations are sometimes **Hæmorrhagic**; they then contain blood corpuscles along with hæmoglobin in solution. This variety of exudation may be due to carcinoma, to tubercle, or to scurvy.

Another variety of exudation is the **Sero-purulent**, in which the fluid is more turbid and the cell elements are more numerous than in the serous form.

In a more advanced stage of inflammation the effusion may be **Purulent**, as in empyema, purulent pericarditis and peritonitis.

Pus has a yellowish or greenish-yellow colour; it is turbid, and of varying consistence; the reaction is alkaline, and the specific gravity high. The colour may be altered by admixture with blood. Microscopical examination reveals enormous numbers of pus corpuscles, with a few red corpuscles, epithelial cells, and various forms of micro-organisms.

TRANSUDATIONS.

These are fluids which do not form as the result of inflammation, but as the result of altered or of obstructed circulation.

They may be serous, hæmorrhagic, or, in a few very rare cases, chylous. The fluid may collect in the subcutaneous tissue (œdema or anasarca), in the pleural cavity (hydrothorax), in the pericardium (hydropericardium), in the abdominal cavity (ascites), in the ventricles of the brain (hydrocephalus), in the tunica vaginalis (hydrocele).

Serous transudations are yellowish or greenish-yellow, clear fluids. **Hæmorrhagic** transudations are more or less reddish in colour, generally, however, only of a faint red colour. **Chylous** transudations have a milky appearance.

The reaction of transudations is alkaline. The specific gravity is lower than in inflammatory effusions. They contain fewer cell elements than serous exudations, but these are of the same nature—leucocytes, red corpuscles and endothelial cells. As a rule, transudations do not coagulate spontaneously. Sometimes, when allowed to stand for a long time, coagulation occurs, and a fibrinous clot is formed, especially if the fluid contains blood. If coagulation should occur, it is only after a much longer time, and in a less degree, than is the case with exudations.

Chemically, transudations consist of water and the elements of blood plasma—serum albumin, serum globulin, fibrinogen, blood salts, extractives, and generally sugar, but no peptones. The salts are almost the same as those of the blood. The percentage of albumin is less than in blood serum; it varies in amount—hence the variation in specific gravity. An important difference between transudations and exudations consists in their relative specific gravities, and in the amount of albumin they respectively contain.

The Specific Gravity of transudations varies according to their locality; it is highest in hydrocele fluid, then follow transudations into the pleura, peritoneum, subcutaneous tissues, and ventricles of the brain. Exudations have a higher specific gravity than transudations, but the specific gravity of the fluid has no constant relation to its source.

There is no sharp and constant difference between the specific gravity of transudations and exudations; but, as a rule, the specific gravity of a pure exudation is rarely *below* 1018, while that of a pure transudation is rarely *above* 1012. Thus, if the specific gravity of a fluid obtained from the peritoneal cavity is higher than 1018, it is almost certainly due to peritonitis (exudation); if lower than 1012, to ascites (transudation). If the specific gravity is between 1012 and 1018, the fluid may be either a transudation or an exudation.

The following figures show the difference in the amount of albumin in transudations and exudations :—

In Pure Exudations.		*In Pure Transudations.*	
Pleura higher than .	40 per cent.	Pleura lower than .	25 per cent.
Peritoneum . .	40–45 „	Peritoneum . .	15–20 „
Skin	40 „	Subcutaneous tissue .	10–15 „
Cerebral meninges .	? „	Cerebral meninges .	5–10 „

Cerebro-Spinal Fluid.—In spina bifida and in cases of chronic hydrocephalus, large quantities of fluid collect—in the former in the sac of the swelling, in the latter in the distended brain ventricles. This fluid closely resembles normal cerebro-spinal fluid. It is clear, the specific gravity is low, and the solids amount to 10 to 13 parts per 1000. When boiled it becomes opalescent, and on the addition of acetic acid a flocculent precipitate separates. Sugar or some other reducing agent —possibly pyro-catechin—is also present. The fluid differs from other transudations in containing as a rule no fibrinogen ; no clot of fibrin is formed when the fluid is treated with fibrin ferment.

Examination of the Puncture Fluid for Micro-Organisms is of some value. The results of recent observations show that the majority of exudations, serous or purulent, which contain no bacteria are tubercular in origin.

Most of the primary, idiopathic, non-tuberculous inflammations of the pleura are said to be due to Fränkel's pneumonococcus. Next in importance to this organism are the various pyogenic micrococci, especially the streptococcus pyogenes. Serous exudations in primary pleurisies, in which pyogenic micro-organisms are discovered, have a greater tendency to become purulent than those containing pneumonococci. Pleurisies accompanying or following pneumonia owe their origin mainly to pneumonococci. Pleuritic effusions due to these micro-organisms run a much less severe course than those dependent on pyogenic bacteria, or than those due to the presence of both kinds of organisms.

Pus or other puncture fluid may be examined for the micro-organisms of tubercle, glanders, malignant pustule, actinomycosis, or leprosy (according to the methods described in pathological text-books), when these diseases are suspected. Tubercle bacilli are rarely found in puncture fluids, though the disease may be undoubtedly tubercular. In tropical abscess of the liver peculiar amœboid organisms are found —the *Amœbæ coli.*

CONTENTS OF CYSTS.

Hydatid Cysts.—The puncture fluid obtained from these cysts is opalescent, or clear and colourless, and is therefore at once distinguishable from ascitic fluid. The reaction is alkaline and the specific gravity low—1006 to 1010. The fluid contains no albumin, or only a very small amount, together with a trace of sugar or some substance which reduces Fehling's solution. A large quantity of inorganic salts, especially sodium chloride, and frequently succinic acid, are present. To detect succinic acid, evaporate the fluid to the consistence of a syrup, acidify with hydrochloric acid, and shake up with ether. Pour off the ether, evaporate, and dissolve the residue in water. This solution gives, with perchloride of iron, a rust-coloured floccular or gelatinous precipitate if succinic acid be present (Wesener).

Microscopical examination is of the greatest diagnostic importance. Minute white specks (scolices) can often be detected in the fluid with the unaided eye; under the microscope they present the well-known appearances of the scolices of the Tænia ecchinococcus. The scolices are round or oval bodies with a somewhat constricted neck, bearing a crown of hooklets and four suckers; sometimes the neck and hooklets are retracted into the body of the scolex. Not infrequently no scolices can be detected, but the deposit at the bottom of a glass in which the puncture fluid is allowed to stand is found to contain the characteristic hooklets. Sometimes portions of hydatid membrane (*i.e.*, cyst wall) are found in the fluid, the membrane being distinguished by its transverse striation or laminated appearance and by its granular inner surface.

If suppuration or hæmorrhage into the sac have occurred, the chemical composition of the fluid is altered accordingly. Only the presence of scolices, hooklets, or the laminated membrane is diagnostic.

Hydronephrosis.—The fluid of a hydronephrotic cyst is generally clear and watery in appearance, with a sp. gr. between 1008–1020. It contains traces of albumin, and usually of urea and uric acid, but in old cysts the urinary constituents may be absent, having been absorbed. As urea and uric acid sometimes occur in other cysts, they are only characteristic of hydronephrosis when present in large amount.

To test for urea:—Evaporate the puncture fluid on a water-bath to the consistence of a syrup. Extract with alcohol; filter the extract and again evaporate to a syrup. A little of the residue dissolved in a small quantity of water is placed on a slide and a drop of nitric acid added. Six-sided plates of nitrate of urea crystallise out, and can be easily recognised with the aid of the microscope.

To test for uric acid:—Add a quantity of hydrochloric acid to the

puncture fluid; allow it to stand twelve to twenty-four hours. Crystals of uric acid are deposited, which may be recognised by the naked eye, by microscopical examination, and by the murexide test (see p. 333).

Ovarian Cysts.—The fluid obtained from these cysts is very variable in character. In colour it may be clear yellow, yellowish-green, dark brown, chocolate-coloured, or almost black. It may be watery and clear, or thick, turbid and slimy, or ropy. The sp. gr. varies from 1002–1055; it is generally between 1010–1025. The fluid has little tendency to coagulate; its reaction is alkaline.

Microscopical examination reveals the presence of red blood corpuscles, leucocytes, and epithelial cells of various forms—squamous, columnar, and ciliated, together with cholestrine crystals; colloid masses are sometimes present. The chemical character of the fluid varies considerably, according to the changes which have taken place in the cyst, such as those caused by hæmorrhage or inflammation. The chief chemical constituents are—water, serum albumin, serum globulin, salts and metalbumin. To test for the presence of *metalbumin*, the fluid is feebly acidified with acetic acid, boiled, and then filtered. Other forms of albumin are thereby removed. To the filtrate an excess of alcohol is added, which produces a white flocculent precipitate. The fluid is allowed to stand for twenty-four hours. It is then filtered, and the precipitate, after being squeezed in linen, is suspended in water. The solution is again filtered, and ought to give the following reactions (*v. Jaksch*):—

1. On boiling it becomes turbid, but does not form a precipitate.
2. Acetic acid gives no precipitate.
3. Acetic acid and ferrocyanide of potassium render the fluid thick, and impart to it a yellow tint.
4. On boiling with Millon's reagent the fluid yields a bluish-red colour.
5. With concentrated sulphuric and glacial acetic acids it yields a violet colour.

In **Dermoid Cysts**, hairs, squamous epithelium, fatty matter, cholesterin crystals, and hæmatoidin are found. If the fluid be purulent on the first tapping, the cyst is probably a dermoid cyst. A low sp. gr. and the presence of only a small amount of albumin is said to point to a cyst of the broad ligament.

Pancreatic Cysts.—The fluid from these cysts has a low sp. gr. It contains serum albumin, but no metalbumin, and rarely mucin. Cholesterin is always present, and frequently blood pigment also (according to v. Jaksch, in the form of methæmoglobin).

Fluid from a pancreatic cyst will digest albumin without the addition of an acid, *i.e.*, in an alkaline solution. No other cystic fluid possesses this property. In old and large cysts, however, this power may be very feeble.

CHAPTER XII.

EXAMINATION OF THE NERVOUS SYSTEM.

ANATOMICAL AND PHYSIOLOGICAL INTRODUCTION.

THERE is no other class of diseases the study of which requires such an intimate knowledge of anatomy and physiology as that of diseases of the nervous system. Again and again the student is baffled when attempting to make a diagnosis, not so much through inability to in-

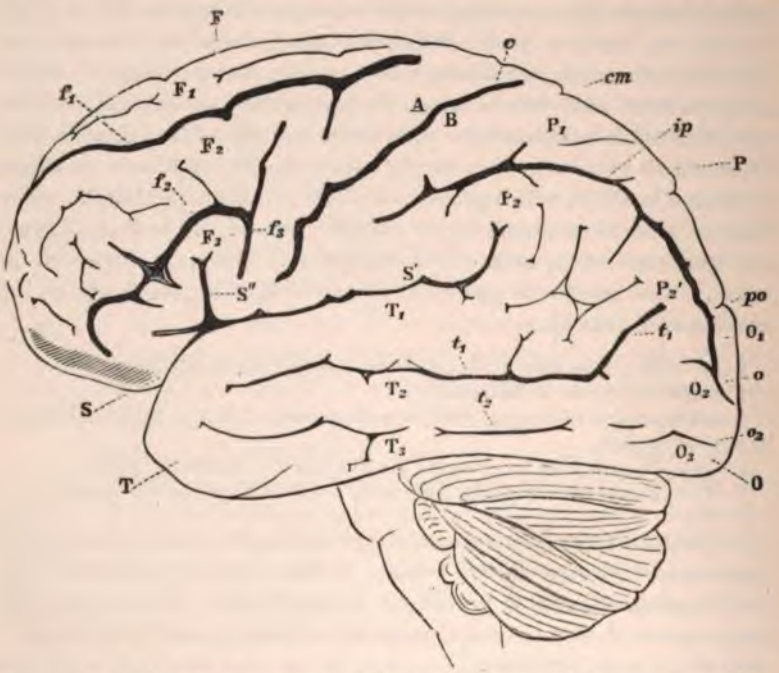


FIG. 199.—Left Side of the Human Brain. F, frontal lobe; P, parietal lobe; O, occipital lobe; T, temporo-sphenoidal lobe; A, ascending frontal, and B, ascending parietal convolution; S, fissure of Sylvius; S', horizontal, S'', ascending ramus of S; C, fissure of Rolando; the other convolutions and fissures are marked with capitals and small letters respectively. (*Ecker.*)

vestigate a nervous case, but because he forgets the function of a particular centre, or the origin and relations of a particular cranial or spinal nerve. It is, therefore, desirable, before entering upon the investigation of symptoms, to briefly review some of the more essential facts relating to the structure and functions of the nervous system,

giving special prominence to those which most commonly call for consideration in the diagnosis of nervous diseases.

Cerebral Convolutions and Fissures.—These are shown and designated in the accompanying diagrams. The three most important fissures are:—(1.) **The fissure of Sylvius**, which divides into a short anterior and a long posterior limb; (2.) **the fissure of Rolando** or the **central sulcus**, which extends from just above the Sylvian fissure to the upper edge of the hemisphere, and separates the frontal from the parietal lobe; (3.) **the parieto-occipital fissure**, which occupies chiefly the median surface, and marks the boundary between the parietal and occipital lobes. On the median aspect it joins the calcarine fissure,

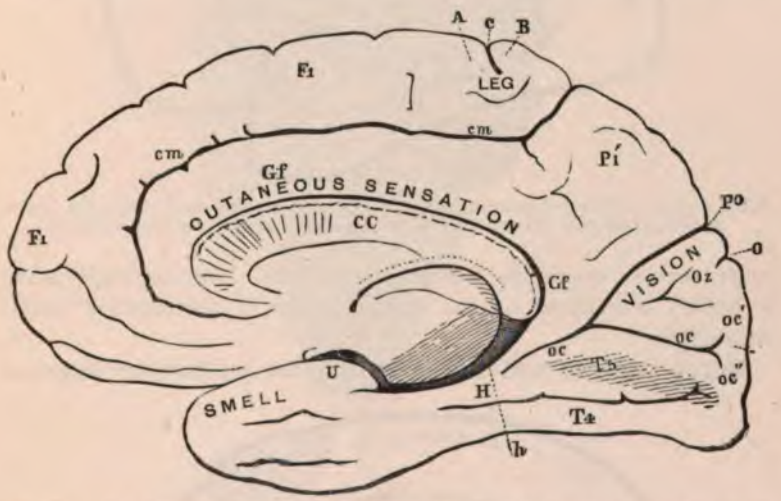
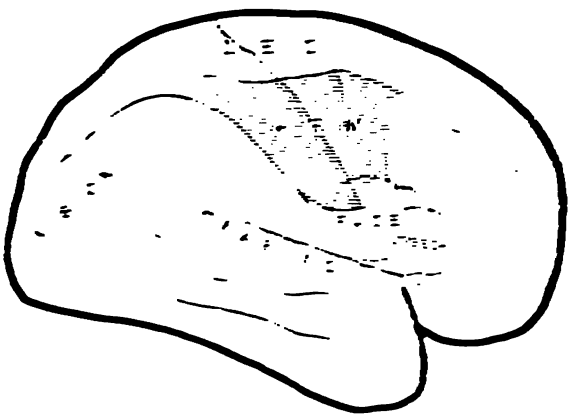


FIG. 200.—Median Aspect of the Right Hemisphere. *CC*, corpus callosum; *Gf*, gyrus fornicatus; *H*, gyrus hippocampi; *h*, sulcus hippocampi; *U*, uncinate gyrus; *cm*, calloso-marginal fissure; F_1, first frontal convolution; *c*, terminal portion of fissure of Rolando; *A*, ascending frontal, and *B*, ascending parietal convolution; *Pi*, quadrate lobule; *Oz*, cuneus; *po*, parieto-occipital fissure; *oc*, calcarine fissure. (*Ecker.*)

enclosing a wedge-shaped area called the cuneus. On the inner surface is also seen the long **calloso-marginal fissure**, the posterior end of which reaches the edge of the hemisphere a little behind the fissure of Rolando. The **precuneus** or **quadrate lobule** lies between the calloso-marginal and the parieto-occipital fissures. The ascending frontal and ascending parietal convolutions, sometimes called the anterior and posterior **central convolutions**, unite below the lower end of the fissure of Rolando, and their lower ends, together with the posterior end of the third frontal convolution, constitute what is known as the **operculum**, which overlies the island of Reil. The upper part of the ascending parietal convolution blends with the superior parietal lobule.

[illegible] the prolongations of the two central convolutions on the median surface of the hemisphere the term paracentral lobule is applied.

In front of the ascending frontal convolution are the three anterior or frontal convolutions which, together with the corresponding



[illegible]

inner surface constitute the prefrontal lobe. The marginal gyrus is mainly constituted by the medial aspect of the superior frontal convolution.

Motor Centres and Motor Path.—The [illegible] region of

[illegible]

the cortex consists of the two central convolutions with the adjacent portion of the adjacent parietal lobule together with the paracentral lobule and part of the quadrate lobule or precuneus. The leg centre occupies chiefly the upper third, the arm the middle third, [illegible]

convolutions, while the **face, lips** and **tongue** are represented in the lowest third of the ascending frontal convolution. The motor centre for **speech** occupies the posterior end of the third frontal convolution, together with the lowest portion of the ascending frontal in the left hemisphere, and also in all probability the underlying island of Reil. It is probable that the centre for the movements of the **head and eyes**

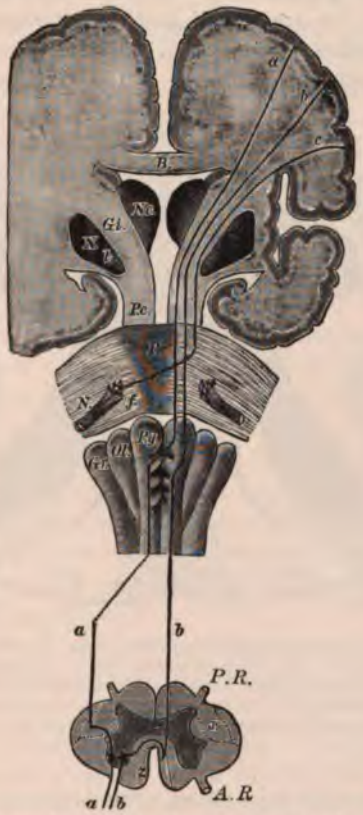


FIG. 203.—Course of the Fibres for Voluntary Movement. *a*, *b*, path for the motor fibres for the limbs and trunk; *c*, fibres for the facial nerve; *Nc*, nucleus caudatus; *Gi*, internal capsule; *Nl*, lenticular nucleus; *P*, pons; *Nf*, origin of the facial; *Py*, pyramids and their decussation; *Ol*, olive; *Gr*, restiform body; *P.R*, posterior root; *A.R*, anterior root; *x*, crossed, and *z*, direct pyramidal tracts. (*Stirling.*)

occupies part of the first and second frontal; that the **trunk muscles** are represented in the median aspect of the ascending frontal; and that in the lower parietal lobule—that is, the lower portion of the parietal lobe—is a centre for the movements of the **upper eyelid**.

Nerve fibres pass down from these motor centres to connect them with the spinal cord. Passing through the white substance of the

hemisphere, they converge to the internal capsule occupying its "knee," and the anterior two-thirds of its posterior segment; the leg fibres are the furthest back, and are next to the sensory fibres. In the crus the motor fibres occupy the middle two-fifths of the crusta. The fibres for the face, and those destined for the tongue, part from the other motor fibres in the pons, and cross the middle line to reach the nuclei of the facial and hypoglossal nerves. In the pons the limb and trunk fibres lie between the superficial and deep transverse fibres, in the medulla they constitute the anterior pyramid, the greater proportion of the fibres of which crosses over at the decussation to run in the

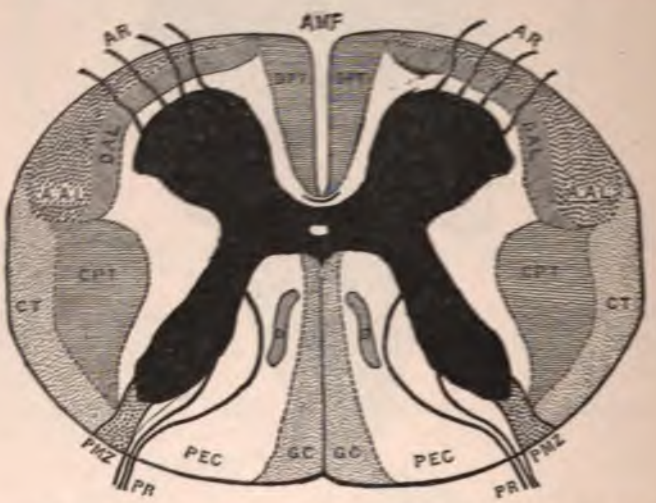


FIG. 204.—Scheme showing the Degeneration-Tracts, and the Paths that do not undergo Degeneration in the Cord. AMF, anterior median fissure; DPT and CPT, direct and crossed pyramidal tracts; AR and PR, anterior and posterior roots; AAL and DAL, ascending and descending antero-lateral tracts; CT, cerebellar tract; D, comma-shaped tract; PMZ, posterior marginal zone; PEC, postero-external column. The parts left white do not undergo degeneration. (*Stirling.*)

lateral column, forming the crossed pyramidal tract. A small though variable proportion of fibres is continued into the anterior column of the same side of the spinal cord, and probably also a few into the lateral tract of the same side. The relative positions of the face and limb centres and fibres are indicated in the diagrams.

In the **cord** the lateral or crossed pyramidal tract is situated in the posterior half of the lateral column, and extends down, gradually diminishing in size, to the end of the cord. The anterior or direct pyramidal tract ("column of Türck") descends in the part of the anterior column adjacent to the median fissure, and usually ceases about the middle of the dorsal region.

The fibres of the pyramidal tract end in grey matter near the motor cells in the anterior horns. From these ganglionic cells spring the anterior roots, which are composed of motor fibres destined for the muscles. In a similar manner the tracts for the motor cranial nerves separate from the pyramidal tract in the crus, pons and medulla, and cross to end near the ganglionic cells, which constitute their nuclei in the floor of the fourth ventricle; and from these cells issue fibres which are collected together to form the motor cranial nerves.

The cortical motor areas are the centres for voluntary movement. The anterior horns of the cord and the corresponding nuclei in the medulla and pons transmit voluntary motor impulses to the peripheral nerves; they are also centres for reflex action. The cortical centres preside over the nutrition of the pyramidal tract, the bulbar and spinal nuclei over that of the motor fibres in the peripheral nerves. A division of the motor path into **two parts**, an upper and a lower part, is therefore a natural one from a physiological point of view, and is very convenient clinically. The **upper** division extends from the cortical centres along the pyramidal tract to the bulbar nuclei and to the anterior horns of the spinal cord; while the **lower** includes the bulbar nuclei and the anterior horns, together with the motor fibres which extend from them to the muscles.

Affections of the Upper Segment.—A destructive lesion of any part of the upper segment gives rise to a **spastic paralysis**, that is, to a loss of muscular power associated with rigidity or spasm of muscles; the pyramidal fibres below the lesion undergo degeneration, but the grey nuclei of the medulla and pons, the anterior horns, motor nerves and muscles do not degenerate.

The situation of the lesion is indicated more or less definitely by peculiarities in the distribution of the paralysis, of which the following are the chief:—

1. If the lesion is **above the decussation** of the pyramids, the limbs and trunk muscles are paralysed on the opposite side of the body. (*a.*) If it is situated above the middle of the pons, the opposite side of the face may be also paralysed. (*b.*) If below the middle of the pons, the face is paralysed on the same side as, but the limbs on the opposite side to, the lesion; this is called "crossed paralysis" or alternate hemiplegia. (*c.*) When the lesion involves the anterior pyramid of the medulla, the face remains unaffected. (*d.*) Irritative lesions of the cortical centres cause convulsions, destructive lesions paralysis on the opposite side; and owing to the divergence of the motor fibres as they approach the cortex, and the consequent separation of those belonging to the face, arm and leg respectively, the paralysis is often limited to the face or to one limb; it is then called monoplegia. The convulsions,

too, of an irritative lesion are usually at first limited to a few muscles of the opposite side of the face or of the opposite limb.

2. If the lesion is **below the decussation** of the pyramids, that is, is situated in some part of the pyramidal tract in the spinal cord, the limbs and trunk muscles are paralysed on the same side. As a rule, in the cord both tracts are affected, and hence both sides of the body are paralysed; in such cases the grey matter rarely escapes injury, and so modifications of sensation or of the reflexes usually accompany a spastic paralysis of spinal origin.

Affections of the Lower Segment, that is, the bulbar or spinal motor nerve-cells or the motor nerve-fibres, produce **atrophic paralysis**; the cells and the fibres below the lesion degenerate, the muscles, supplied by the affected nerves, also undergo a rapid atrophy, and give degenerative reactions to electricity (see p. 395).

Sensory or Centripetal Path.—Cutaneous sensations, received by the terminal apparatus in the skin of the trunk and extremities and from the mucous membranes, are conveyed along sensory nerve-fibres to the posterior roots of the spinal nerves. The position of the sensory path, however, between the posterior roots and the cortex of the brain is for the most part uncertain. Gowers believes that sensibility to pain (and with it possibly also that to temperature) is conducted by the antero-lateral ascending tract, while sensibility to touch ascends the posterior column. Passing through the posterior half of the medulla and pons, where the path from the fifth nerve joins it, the sensory path goes through the crus cerebri and enters the posterior third of the hinder limb of the internal capsule; this part, which receives also fibres conveying sensory impulses of taste, hearing, smell and vision from the opposite side, is called the "**sensory crossway.**" Higher, the sensory fibres pass into the white substance of the hemisphere, and those conveying impulses from the cutaneous surface of the body end, in all probability, in the cortex of the central convolutions and of the parietal lobe.

Gowers considers it probable that muscular sensibility is transmitted upward along the same side of the cord, possibly in the posterior median column, whereas all other forms of sensibility immediately cross to the opposite side of the cord. Ferrier, however, says "the evidence is in favour of the view that the *whole* of the sensory paths pass up the opposite side of the spinal cord, and that they are not contained either in the posterior median columns or in the direct cerebellar tract, or in the antero-lateral tract; . . . and we are led to suppose that the sensory tracts ascend in immediate relation with the *central grey matter.*"

A destructive lesion of the parietal portion of the cortex, or of the

hindmost part of the internal capsule, will produce loss of sensation in the skin and mucous membranes of the whole of the opposite half of the body. Also it must be remembered that lesions of the so-called motor area are sometimes attended by sensory disturbance. For example, the extremity of a paralysed limb may have its sensibility blunted or perverted as regards locality; and motor spasms from irritation of this cortical area are often preceded by a sensory aura.

Destruction by injury or disease of one side of the cord produces loss of cutaneous sensibility on the opposite side below the level of the lesion, but according to Gowers, loss of muscular sensibility on the same side.

The Visual Path.—The optic tracts pass from the retinæ along the optic nerves to the chiasma; here there is a partial decussation, the fibres from the inner half of each retina crossing to enter the optic tract of the opposite side, while the fibres derived from the outer half of each retina run along the optic tract on the same side (see Fig. 205). The visual path passes in the optic tract to the anterior pair of the corpora quadrigemina, thence in the posterior segment of the internal capsule by the corpora geniculata, through the white substance of the hemisphere to the cortex of the occipital lobe. But, in addition to this half-vision centre in the occipital lobe, there is, in all probability, a higher visual centre situated in the angular gyrus, in which the whole of the opposite field of vision is represented, and to a less degree the whole field of the eye of the same side.

Destruction of the occipital cortex, especially of the cuneus, or of any portion of the optic tract between the occipital lobe and the chiasma, renders the outer half of the retina on the same side and the inner half of the opposite retina blind to visual impressions; thus if the left optic tract is the seat of a lesion (see B, Fig. 205), the patient, when looking straight before him, cannot see objects situated to his right. It is probable that a lesion of the left angular gyrus leads to extreme concentric diminution of the field of vision of the right eye, and to a moderate diminution of the field of the left eye.

The Auditory Path.—The auditory nerve, directed inwards from the temporal bone, passes between the pons and medulla to its nucleus in the floor of the fourth ventricle. This has important central connections with the cerebellum, and also, by means of the opposite internal capsule at its posterior part, with the auditory centre in the first temporo-sphenoidal convolution.

The Olfactory Path.—Some of the fibres of the olfactory nerve enter the uncinate gyrus of the same side; others cross, perhaps by means of the anterior commissure, to the opposite hemisphere, reaching the cortex through the posterior end of the internal capsule.

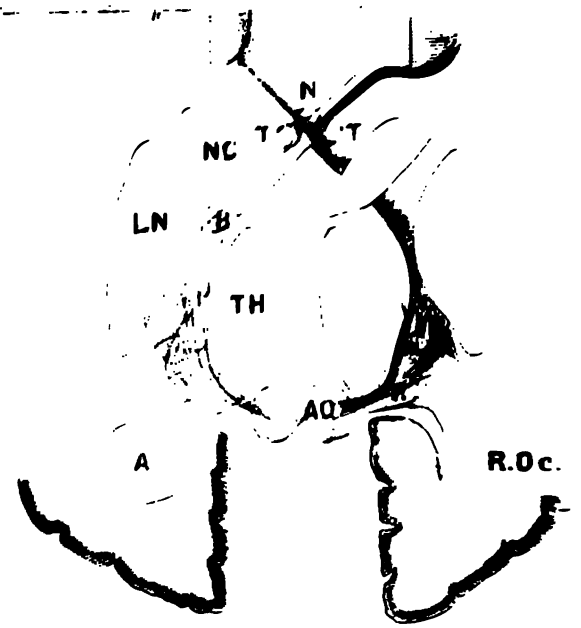


Fig. [illegible] Diagram to illustrate the [illegible] of Vision in Man. (*After Sherrington, modified.*) The right optic tract [illegible] supplies the temporal side of the retina of the right eye and the nasal side of the retina of the left eye; excitation of these parts of the retina produces vision in the [illegible] of the fields of vision. The right optic tract is represented as ending in G.L. the lateral [illegible] geniculate body; it is the pulvinar and in A, the anterior corpus quadrigeminum. Connecting these bodies with the right occipital lobe [illegible] is the optic radiation represented by three lines; N the nucleus caudatus, LN the nucleus lentiformis and TH the optic thalamus. A lesion 1 or a lesion 2 would produce *right hemianopsia*; a lesion 3 would produce *temporal hemianopsia* while the lesions 4 or 5 would produce *nasal hemianopsia* [illegible]

Psychical Centres.—It is believed that the seat of the higher mental processes is located in that portion of the cortex which lies in front of the motor areas. Lesions of this part, that is, of the prefrontal lobes, may produce considerable mental changes; but it is a

less true that wide-spread disease of other portions of the cortex leads to dulness of the higher faculties, and it is probable that mental operations are subserved by very extensive areas of the cortical grey matter.

The Centre and Paths for the Co-ordination of Movements.—The paths in the nervous system which appear to be related to the accurate adjustment of muscular action are closely connected, and as it were interwoven with, those which conduct certain sensory impulses; it was therefore fitting to consider the latter first. The subject is a complex one, and few, if any, positive assertions can yet be made with regard to it. There are, however, a few facts relating to the cerebellum and to certain tracts in the spinal cord which require a brief notice. The **Cerebellum** consists of two lateral hemispheres and a middle lobe, and by means of its peduncles it is intimately connected with the cerebrum, pons and medulla oblongata. Its connection with the cerebral hemispheres is mainly a crossed one, fibres passing from the prefrontal, temporal and occipital lobes to the cerebellar hemisphere of the opposite side. The middle lobe of the cerebellum is largely concerned with the maintenance of equilibrium, and may be regarded as the centre for the co-ordination of muscular contractions. It acts, however, in strict subordination to the cerebrum, and may be said to regulate muscular contractions, which are initiated, and are subject to constant changes, by the action of the higher centres in the cerebrum.

Lesions of the middle lobe produce a staggering gait; when situated in its upper part, there is a tendency to fall forwards; when in its lower part, to fall backwards. Lesions limited to one of the cerebellar hemispheres are frequently unattended by definite symptoms, but the patient may exhibit a tendency to fall towards the affected side, or there may be a forced rotatory movement towards this side. But the latter symptom is usually due to active disease of the middle peduncle of the cerebellum.

The **middle lobe** of the cerebellum is connected with the periphery of the body by several important tracts. Two are situated in the spinal cord, namely, the posterior median or Goll's column, and the direct cerebellar tract. Both undergo an ascending degeneration, when the fibres of which they are composed are interrupted. The fibres of each tract are derived from the posterior roots, those of the direct cerebellar tract chiefly from the posterior roots of the upper dorsal and cervical regions, and not from those of the lower extremities; this tract has an intimate connection with the posterior vesicular column. It is probable that both the columns of Goll and the direct cerebellar tracts consist of cerebello-afferent fibres, and it may be pretty confidently stated that they conduct sensory impressions from the muscles to the cerebellum.

Interruption or disease of these tracts, or of the posterior roots from which they are derived, appears to be one of the principal factors in the production of incoördination of movement—a characteristic feature of locomotor ataxia.

A third centripetal path is constituted by the auditory nerve, especially that part of it which receives impressions from the semicircular canals, these impressions being conveyed to the auditory nuclei, and thence to the cerebellum. Irritation of the nerve-fibres to the canals produces a feeling of giddiness and incoördination of muscular movements, so great sometimes that the patient finds it difficult or impossible to stand. These, together with aural symptoms, are the chief phenomena in Menière's disease or auditory vertigo.

It is also highly probable that the cerebellum is indirectly connected with the periphery by a fourth tract, namely, the visual; for our relations to external objects are largely estimated by sight and by the position of the eyes. It may indeed be assumed that centripetal fibres pass from the centres for the movements of the eyeballs to the middle lobe of the cerebellum, which regulates the attitudes of the body, so far as they relate to the maintenance of equilibrium.

The Central Ganglia.—The optic thalamus is connected by fibres with the tegmentum of the crus, with the superior peduncle of the cerebellum, in all probability with the optic nerves, and with all parts of the cerebral cortex. It is probable that the thalamus has to do with some of the higher reflex processes, but lesions limited to it, and not involving the adjacent internal capsule, do not produce any symptoms, with the exception possibly of tremors or clonic spasms, involving movements on the opposite side of the body.

The nuclei of the corpus striatum have no connection with the cortex, nor with the pyramidal motor path; but both the caudate and the lenticular nuclei have extensive connections with the cerebellar hemisphere of the opposite side. Destruction of the corpus striatum may be unattended by symptoms.

The Relative Position of the Nerve Nuclei beneath the floor of the fourth ventricle and the Sylvian aqueduct is shown in the accompanying diagrams. It is important to observe: (1) that the nuclei of the hypoglossal and spinal accessory nerves are close together, and that injury to their roots paralyses the tongue, palate and vocal cord on the same side. Such paralysis is also caused by degeneration of the nuclei; then the lips also suffer, probably because the fibres of the facial nerve which supply the orbicularis oris dip down to the hypoglossal nucleus, and thus we have "bulbar" or "labio-glosso-laryngeal-paralysis." (2.) That although the facial nerve winds round the nucleus of the sixth nerve, it has no real connection with it.

(3.) That the nucleus of the third nerve is made up of three parts —an anterior centre for accommodation, another for the light reflex of the iris, and a third, the largest, representing all the other eye muscles supplied by the third nerve. (4.) That the nucleus of the fourth nerve is continuous with that of the lowest part of the third. Some fibres,

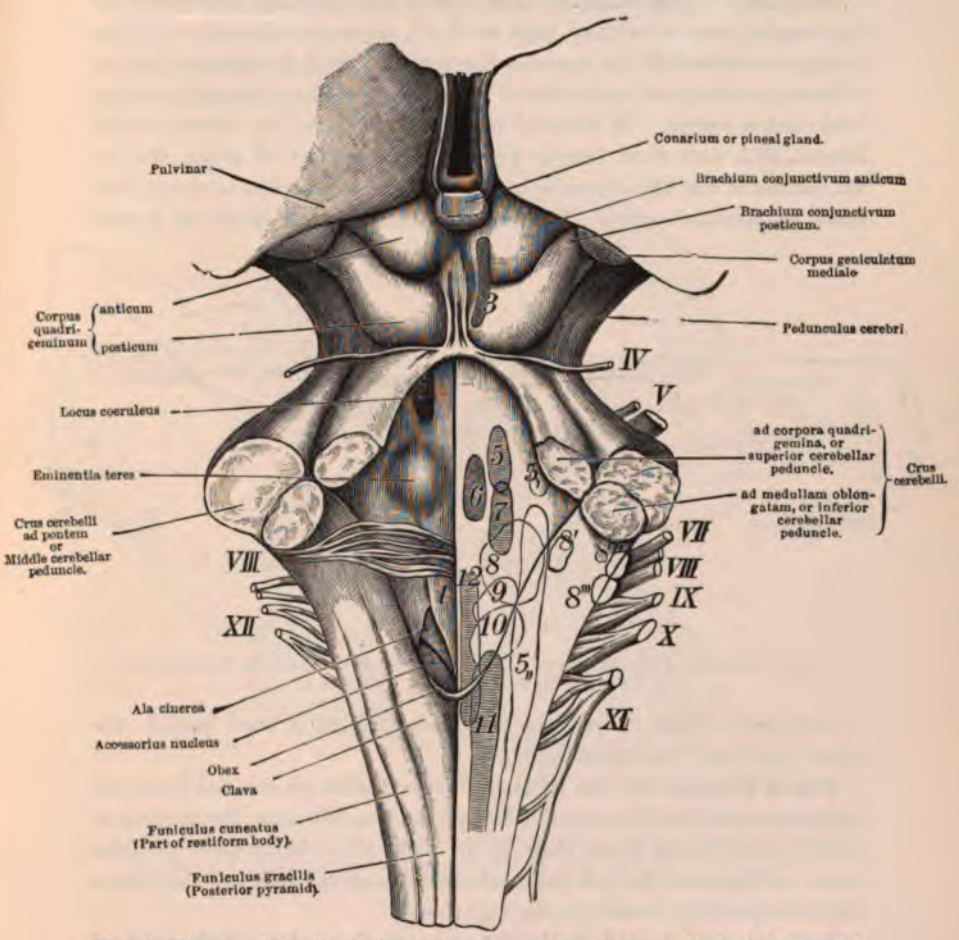


FIG. 206.—Diagram of the Fourth Ventricle. The numbers IV.–XII. indicate the superficial origins of the cranial nerves, while 3–12 indicate their deep origin, *i.e.*, the positions of their central nuclei; *t*, funiculus teres. (*Landois and Stirling.*)

however, probably come from the nucleus of the sixth. Indeed, the connection between the nuclei of these three nerves, which govern the complex movements of the eyes, is a very close one. (5.) That the deep origin of the fifth nerve is a very extensive one: it reaches from beneath the corpora quadrigemina down to the grey matter of the spinal

and, being close to the basal gelatinous, and to the origin of the cervical nerves below. (6.) That the sixth nerve having a longer course than any of the cranial nerves before they enter the dura mater, are most exposed to injury, and are especially liable to be damaged as they pass beneath the pons by anything pressing upon it.

Lesions at the Base of the Brain are liable to injure some of the cranial nerves as they pass to their respective foramina. Thus a tumour situated in the anterior fossa of the skull is apt to affect the olfactory nerves, and by backward extension to damage the optic or the oculo-motor nerves. If situated in the middle fossa, the second, third, fourth, fifth and sixth nerves are likely to suffer: of these, the involvement of the fifth nerve or of the Gasserian ganglion is usually the most prominent feature. If situated in the posterior fossa, the fourth

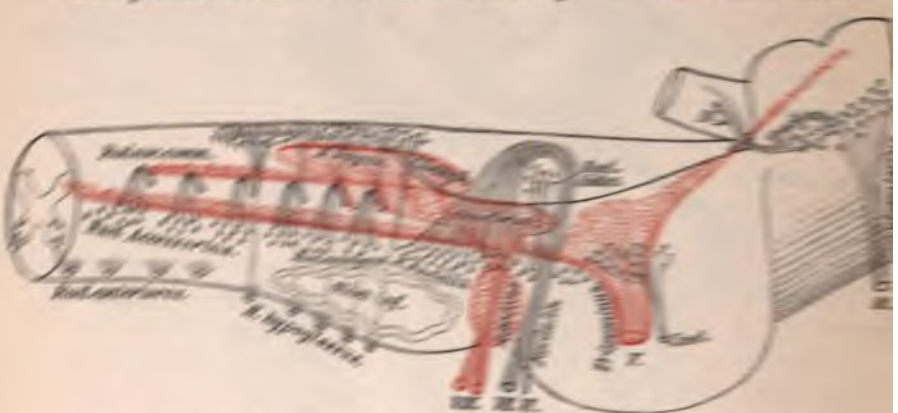


FIG. 107.—Scheme of the Disposition of the Nuclei of Origin of the Cranial Nerves in the Region of the Bulb and Pons. (Hilleger.)

or any nerve below it may be involved, and, at a later period, the motor tract may become affected.

Blood-Vessels of the Brain.—The **arteries** are derived from the internal carotid and vertebral arteries; on the left side the current of blood is much more direct than on the right side; hence solid particles more readily enter the left internal carotid and the left vertebral than the corresponding vessels on the right side.

Each internal carotid divides into an anterior and a middle cerebral artery; these, together with the posterior cerebral and the communicating arteries, form the circle of Willis.

Cortical Branches.—The anterior cerebral arteries curve round the corpus callosum, and supply on the outer surface the first and second frontal and the top of the ascending frontal convolutions, also the inner surfaces of the hemispheres in front of the parieto-occipital fissure, together with portions of the orbital lobules. The posterior

cerebral arteries supply the lower aspect of the temporal and the occipital lobes. The middle cerebral supply the rest of the cortex, and hence all the motor region, with the exception of portions of the leg centres, which receive blood from the anterior cerebral vessels. The middle cerebral also supply the sensory portions of the cortex, together with the auditory and speech centres, and probably the higher visual centres, the half-vision centres receiving blood from the posterior cerebrals, which supply also the sensory part of the internal capsule and the corpora quadrigemina.

FIG. 208.

FIG. 209.

FIGS. 208 and 209.—Areas of the Cortex supplied by Branches of the Cerebral Arteries. The areas shaded with horizontal lines represent the distribution of the anterior cerebral artery; the areas shaded with vertical lines the distribution of the posterior cerebral artery. The unshaded area in Fig. 208 represents the distribution of the middle cerebral artery.

Central Arteries arise from the three cerebrals near their origins, and also from the circle of Willis. There are six groups, two mesial and four lateral, two on each side.

The small anterior mesial group from the anterior cerebrals supplies the head of the caudate nucleus; the small posterior mesial group from the posterior cerebrals supplies the inner part of the optic thalamus and the walls of the third ventricle. Hæmorrhage from rupture of these small branches of the anterior and posterior cerebral arteries is

apt to burst into the ventricles. The internal capsule and the central ganglia are mainly supplied by the antero-lateral groups, which consist of small arteries derived from the commencement of the middle cerebrals. There are internal branches which penetrate the inner portions of the lenticular nucleus and internal capsule, and external branches, which are larger, and are divisible into two sets—an anterior set, named **lenticulo-striate arteries**, and a posterior set, the **lenticulo-optic arteries**. Both these sets, but especially certain branches of the former set, are particularly liable to rupture. The arteries of the

FIG. 210.—Transverse Section of a Cerebral Hemisphere. CCα, corpus callosum; NC, caudate nucleus; NL, lenticular nucleus; IC, internal capsule; CA, internal carotid artery; αSL, lenticulo-striate artery ("artery of hæmorrhage"); F, A, L, T, position of motor areas governing the movements of the face, arm, leg and trunk muscles of the opposite side. (*Horsley.*)

posterior lateral groups which spring from the posterior cerebrals supply the posterior ends of the optic thalami, and their rupture usually damages the posterior third or sensory portion of the internal capsule. The crura and corpora quadrigemina are also supplied by the posterior cerebrals.

Between the central and cortical systems there are no anastomoses, nor do the central branches communicate with one another; but anastomoses often exist between the cortical branches.

The pons and medulla receive median and lateral branches from the vertebral, basilar and cerebellar arteries. There is no communication

between them, and hence softening is not uncommon; but it is rare in the cerebellum, because the superior, middle and inferior cerebellar arteries freely communicate with one another.

The Veins.—The leading anatomical points to be remembered in connection with the venous circulation of the brain are:—

1. That the veins from the greater part of the cortex pass upwards and forwards into the superior longitudinal sinus, the direction of the current of blood being opposed to that in the sinus. This is one of the chief reasons why clots are readily formed in the sinuses and cortical veins.

2. That the veins of Galen, which receive blood from the lateral ventricles, empty themselves into the straight sinus. Any obstruction of these veins, as by a tumour of the middle lobe of the cerebellum, causes effusion into the ventricles, and constitutes a factor in the production of hydrocephalus; at the same time it is doubtful whether much hydrocephalus can be present unless the communication between the ventricular cavity and the subarachnoid space (by means of the foramen of Magendie) is completely occluded.

3. That nearly all the blood from the cerebrum and cerebellum is conveyed directly or indirectly into the lateral sinuses, and thence to the internal jugular veins.

4. That there are several important communications between the intra- and extra-cranial veins. Thus the veins of the nose and most of those of the scalp communicate with the superior longitudinal sinus; the occipital veins with the lateral sinus by means of the mastoid veins; the deep cervical veins with the inferior petrosal sinus; while a communication is established between the facial vein and the cavernous sinus by means of the ophthalmic vein.

Blood-Vessels of the Spinal Cord.—Two points of practical importance in connection with the distribution of the arteries may be mentioned.

1. Owing to their tortuous course before entering the cord, they are much less exposed to pressure than those going to the brain, and hence hæmorrhage into the cord, from degeneration and rupture of the arterial wall, is a rare event.

2. The arteries which supply the cervical and upper dorsal regions pass almost horizontally to the cord, but many of those which nourish the lower end of the cord have a long ascending course, the arteries accompanying the nerves of the cauda equina being often several inches in length. This, together with the smallness of their calibre, offers much resistance to the blood-stream, and may possibly explain to some extent the proneness of the lower part of the cord to become diseased.

Relations of the Spinal Cord and of the Spinal Nerves to the Vertebral Spines.—[illegible]

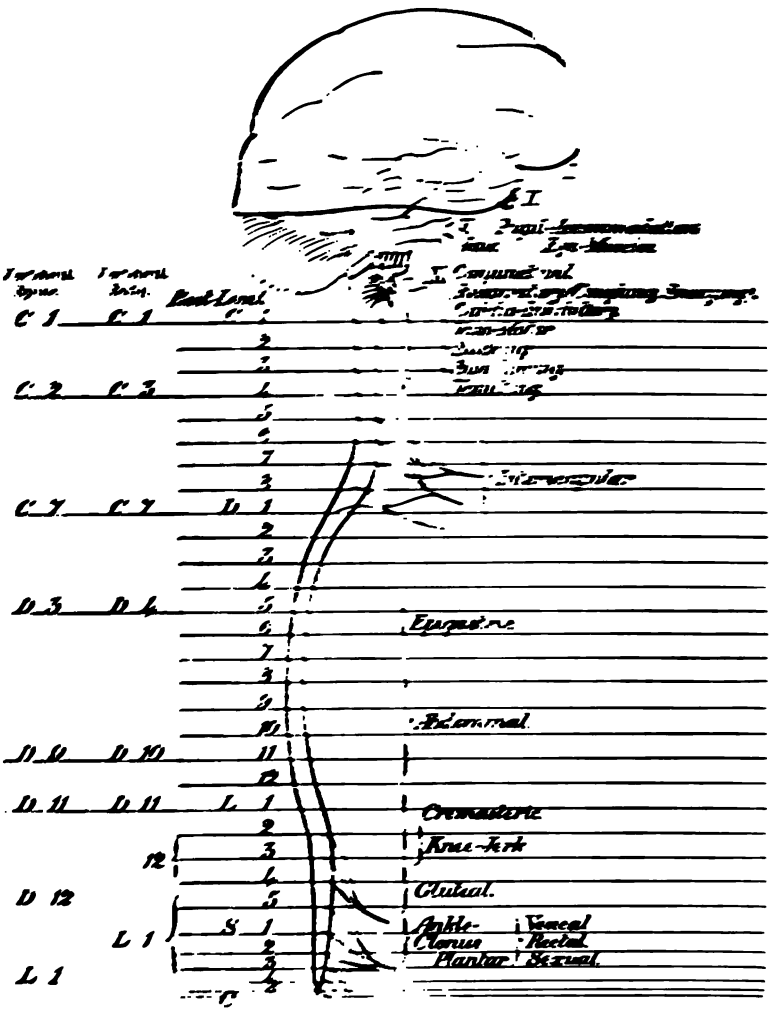


Fig. [illegible] Diagram and Table of Reflex Actions, showing the Place in the Cord through which the Reflex occurs, and its level with regard to both the Bodies and the Spines of the Vertebræ (Hall.)

reaches as low as the third lumbar vertebra, but in the adult it is opposite the lower border of the first lumbar vertebra.

The cervical enlargement extends from the upper end of the cord to the first or second dorsal vertebra, and corresponds to the bodies and

Relations of the Spinal Cord and of the Spinal Nerves to the Vertebral Spines.—At birth the lower end of the cord

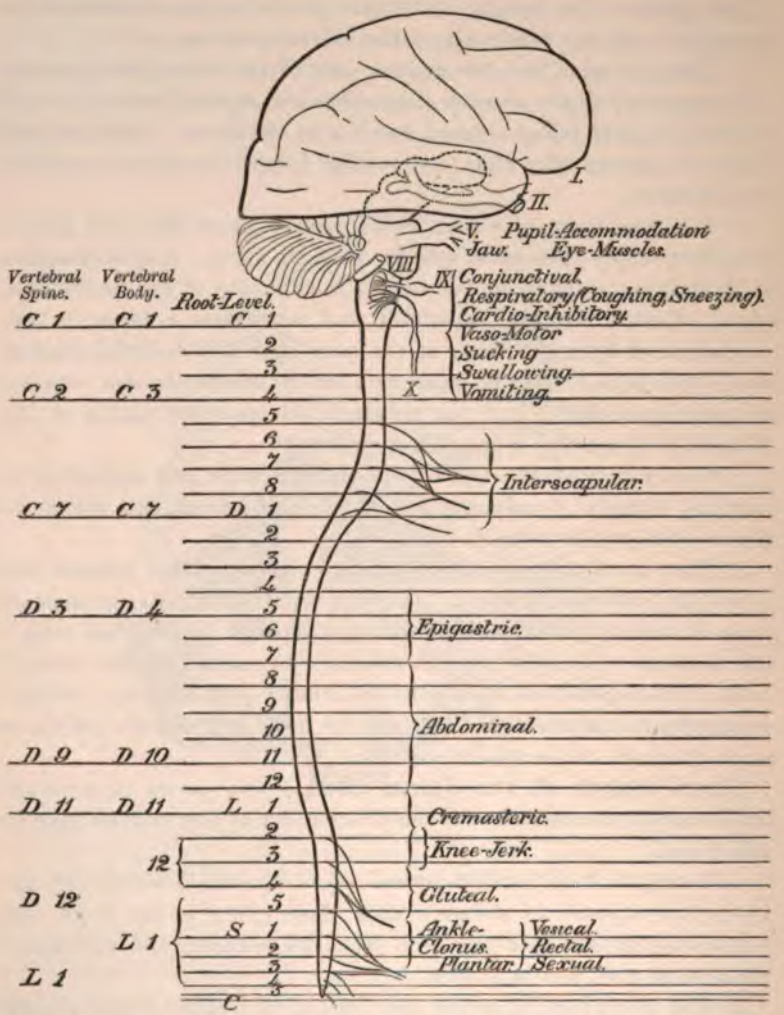


FIG. 211.—Diagram and Table of Reflex Actions, showing the Plane in the Cord through which the Reflex occurs, and its level with regard to both the Bodies and the Spines of the Vertebræ. (*Hill.*)

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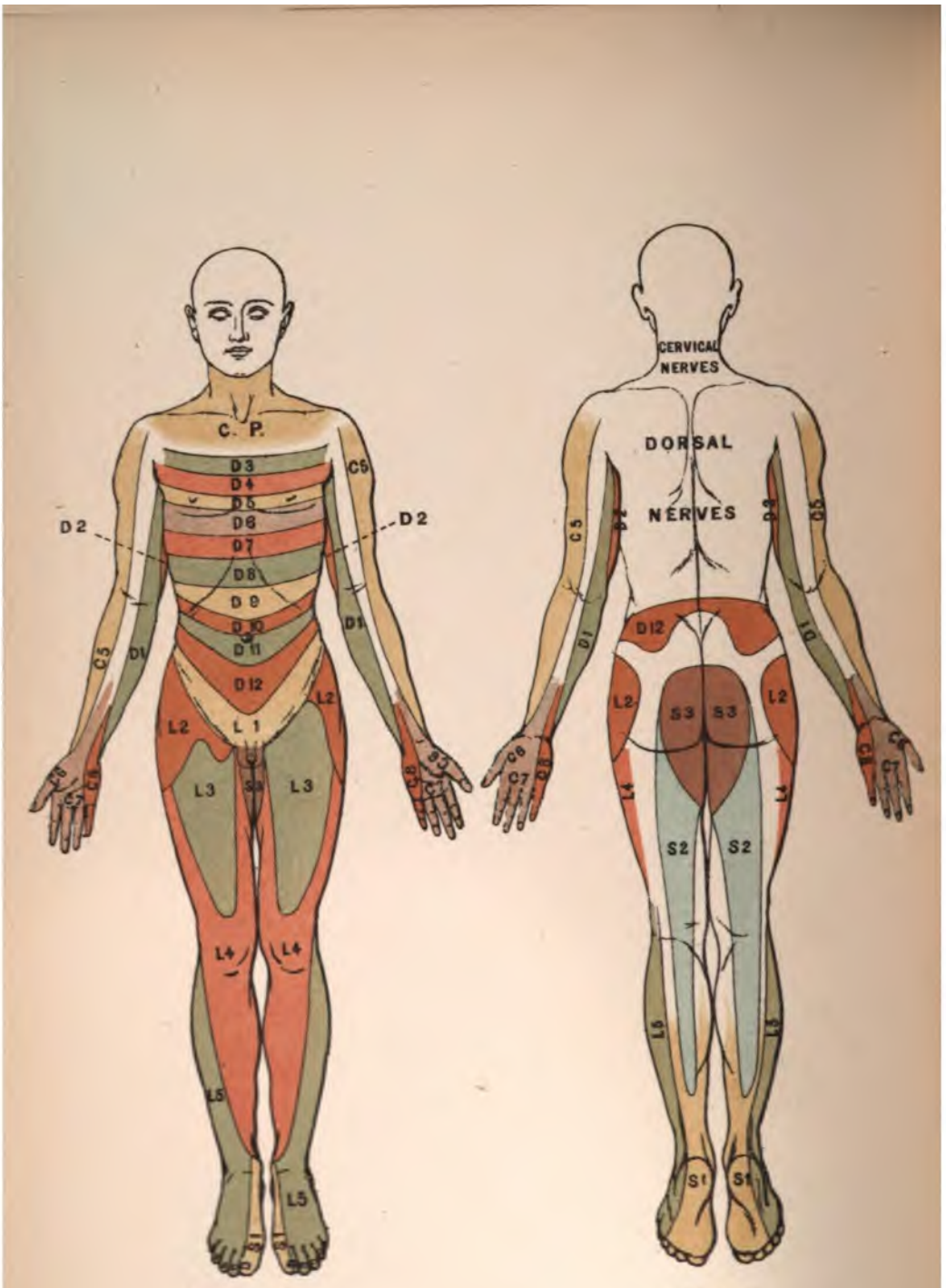


Plate I. Plate II.

Diagrammatic view of the sensory distribution of spinal nerve-roots.

spines of the cervical vertebræ. The lumbar enlargement corresponds to the lowest three dorsal and the first lumbar spines.

The spinal nerves do not leave the cord at the vertebræ corresponding to them in number; and inasmuch as disease of the cord or its roots is frequently produced by lesions of the vertebræ or of the spinal membranes, it is impossible to make an accurate diagnosis of the seat of the lesion without a knowledge of the relations between the roots and the vertebral spines. These relations, as well as the various levels at which the reflexes occur, are shown in Fig. 211.

Distribution of the Spinal Roots.—This, so far as it has been ascertained by the labours of Ferrier, Thorburn and others, is indicated in the following table. The facts given in the column relating to the sensory distribution of the spinal nerve roots are also shown, diagrammatically, in Plates I. and II. (modified from Thorburn, "Brain," 1893). "It must, however, be fully understood that the boundaries of the areas represented are not to be taken as absolutely defined; they are merely general delineations of the usual distribution of each root;" for example, in the case of the sole of the foot, there is no satisfactory evidence as to the true boundary between the first sacral and the fifth lumbar.

Relation of Motor and Sensory Functions to Spinal Roots.

Nerve Roots.	Motor.	Sensory.
C. 1–3	Small rotators of head. Sterno-mastoid. Trapezius (upper part). Levator anguli-scapulæ. Scaleni. Depressors of hyoid bone.	Scalp and neck. In Plate I., CP represents the area supplied by the descending branches of the cervical plexus.
4	Supra- and infra-spinatus. Diaphragm. Teres minor (?).	Neck and upper part of chest.
5	Biceps and brachialis anticus. Deltoid. Supinators longus and brevis.	Over deltoid and outer aspect of arm and forearm, as far as styloid process of radius.
6	Subscapularis. Pronators. Teres major. Latissimus dorsi. Pectoralis major. Triceps. Serratus magnus.	Central portions of anterior and posterior aspects of arm and forearm.
7	Extensors of wrist. Long extensors of fingers.	

RELATION OF MOTOR AND SENSORY FUNCTIONS TO SPINAL ROOTS —*continued.*

Nerve Roots.	Motor.		Sensory.
C. 8	Flexors of wrist. Long flexors of fingers.		Inner side of little finger, of hand, forearm and arm.
D. 1	Interossei. Other intrinsic muscles of hand.		
2–12	Lower part of trapezius. Muscles of chest and abdomen. Erector spinæ.		Chest and abdomen, and upper part of buttock.
L. 1	... ...		Ilio-hypogastric and ilio-inguinal.
2	Cremaster.		Outer and upper part of thigh. The areas are represented differently on the two sides in Plate I., in order to illustrate probable variations in the distribution of the second lumbar.
3	Sartorius. Adductor and flexors of thigh.		Anterior aspect of thigh.
4	Extensor of knee. Abductors of thigh.		Anterior and inner part of leg; also inner and outer portions of thigh.
5	Hamstring muscles.		Outer aspect of leg and foot; also probably part of back of thigh.
S. 1 2	Calf muscles and glutei. Peronei. Flexors of ankle. Intrinsic muscles of foot.	Nervi erigentes.	A narrow strip on back of thigh, back of leg and ankle, sole; inner part of dorsum of foot.
3	Perineal muscles. Penis muscles.		Perineum, external genitals, "saddle-shaped" area of back of thigh.
4	Bladder and rectum.		Skin from coccyx to anus.

CLINICAL EXAMINATION, OR THE INVESTIGATION OF THE SYMPTOMS PRODUCED BY DISEASE OF THE NERVOUS SYSTEM.

The symptoms to be considered in the present section may be conveniently grouped under the following headings:—

1. Disorders of muscular action.
2. Disorders of sensation.
3. Disorders of reflex action.
4. Disorders of language.
5. Disorders of the special senses.

DISORDERS OF MUSCULAR ACTION.

Muscular action may be increased or diminished in strength, or it may be perverted, without presenting any abnormality as regards strength.

Increased Muscular Action.—Spasm is a term applied to increased and involuntary muscular action, that is, to muscular contractions which occur independently of any voluntary stimulus. It may affect the involuntary or the voluntary muscles of the body, and be of the clonic or tonic variety. When affecting the voluntary muscles, spasm indicates undue excitability of some portion of the motor tract, or of the muscles themselves, in consequence either of morbid irritability or of defective control of the reflex centres.

Tonic Spasm.—This means uninterrupted muscular contraction, the duration of which may vary from a few minutes to many months. Persistent tonic spasm is often termed *rigidity*, or, if limited to a particular group of muscles, *contracture*. The latter condition may result from long-continued irritation of motor roots; or it may affect healthy muscles whose opponents are paralysed—as, for example, in infantile paralysis, where the contraction and shortening of the healthy muscles of the affected limb are the results of adaptation to posture, and constitute an important factor in the production of the various deformities met with in that disease.

According to the presence or absence of paralysis, cases of tonic spasm may be broadly separated into two groups.

Group 1.—**Tonic Spasm is dominant; Paralysis is absent or inconspicuous.**—Such spasm may affect a single muscle, as the diaphragm; a group of muscles as in tetany; or the majority of the muscles of the body as in tetanus, catalepsy and strychnine poisoning. Tetanic attacks also occur in meningitis, and rarely in tumours of the cerebel-

lum, probably from pressure on the pons. In Thomsen's disease the commencement of a voluntary movement is attended by so much rigidity of the muscles that any intended action is hindered or prevented.

A simple variety of tonic spasm is illustrated by *cramp*. This symptom is prominent in cholera. It also occurs in peripheral neuritis, especially the alcoholic variety; indeed, cramp of the calf-muscles is one of the commonest premonitory symptoms of alcoholic neuritis, and is found in association with coldness, and sensations of numbness and tingling of the hands and feet. Muscular cramps and startings of the limbs are apt to occur with the greatest severity just as the patient is about to fall asleep. Frequently, also, the fingers and toes are attacked and distorted by painful cramps. Further, in

FIG. 212.—Spasm of Interossei in a well-marked case of Alcoholic Paralysis.

the later stages of multiple neuritis, when paralysis is pronounced, irritative motor phenomena are sometimes present. For example, in a case of alcoholic paralysis under the author's care, the position of the hands somewhat resembled that seen in tetany (see Fig. 212), and it was found that the flexion of the first phalanges of the fingers, and the extension of the terminal ones, were mainly caused by spasm of the interossei and lumbricales; this was proved by the firmness with which the fingers were pressed together, and by the resistance experienced on trying to separate them, or to extend them at the metacarpo-phalangeal joints. The fingers, too, were never completely at rest, and their quivering movements appeared to be due to intermittent contractions of the interossei. In such cases it seems likely that the spasms are due to irritation of peripheral nerve-fibres, just as spasm of the ocular muscles may result from basal meningitis causing irritation

of the motor nerve-trunks, or facial spasm from the pressure of a tumour on the facial nerve.

In other examples of tonic spasm, as tetany or torticollis, it is often impossible to say what part of the motor tract is in a morbid state. Some authorities believe that the spasms of tetany depend on disturbance of the cortical motor areas; against this view may be mentioned the persistence of the spasms during sleep and chloroform anæsthesia, and the fact already mentioned (see page 65), that the position of the hand in tetany differs from that seen in the convulsive attacks of infancy. The pathology of many varieties of torticollis is equally obscure. Hence it is particularly important that the student should make careful observations of every case of tonic spasm that comes before him, noting its distribution and degree, and whether the muscles affected with spasm are supplied by the same nerve, or whether they are functionally associated although supplied by different nerves. He should then look for any signs of motor weakness or sensory disturbance in the part affected. Finally, he should endeavour to determine the most probable situation of the lesion, and especially whether it is in the upper or in the lower segment of the motor tract.

When the involuntary muscles are attacked by spasm, it is commonly of the tonic variety—for example, the spasm of the pharynx in hydrophobia, of the glottis in laryngismus stridulus, of the intestinal wall in certain cases of colic.

Group 2.—**Tonic Spasm and Paralysis are Associated in various Proportions.**—The regional pathology of this group is much clearer than that of cases where tonic spasms are the only morbid phenomena present. As a rule, the combination of paralysis and spasm indicates a lesion of some portion of the upper segment of the motor tract (see p. 355). The condition is spoken of as one of spastic weakness or spastic paralysis, and will be more fully described when we come to consider the various forms of paralysis. Let it, however, be remembered that a spastic paralysis may possibly sometimes be due to disease of the lower segment of the motor tract (see Fig. 212).

Clonic Spasm.—Here contraction and relaxation of muscular fibres occur in quick succession. It may affect a single muscle—as, for example, the orbicularis palpebrarum—when twitching movements of the eyelid will be observed; or a group of muscles, as in clonic torticollis, when the head is rotated obliquely to one side by a succession of jerks.

Tremor is the most delicate form of clonic spasm, and between it and the coarsest variety of clonic spasm there is every possible gradation. When limited to individual muscular fibres, it is called **fibrillary contraction.** These contractions are visible as wavy oscillations under

the skin, and constitute one of the earliest signs of muscular atrophy. When their presence is doubtful or inconstant, they may be elicited or increased by sharply tapping over the muscles, or sometimes by cooling the surface of the skin. Such fibrillation of muscle is common in peripheral neuritis, and occurs to a marked degree in progressive muscular atrophy (chronic anterior poliomyelitis), while it is less frequent and severe in infantile paralysis (acute anterior poliomyelitis), and usually absent in the group of myopathic atrophies.

Tremor, when more extensive in distribution, causes trembling of

FIG. 213.—Unilateral Clonic Facial Spasm—*Mimic spasm* or *Convulsive tic.*

the limbs or trunk, and is of many varieties. It may be fine or coarse in character, and jerky or regular in rhythm. It may be constant during waking hours, or occasionally continue during sleep; or it may intermit, and occur only during voluntary movements. For example, in paralysis agitans the tremor persists during repose, although it generally subsides during sleep; but in disseminated sclerosis the tremor is absent during rest, but appears when voluntary movements are performed.

The tremor of **paralysis agitans**, sometimes called *convulsive tremor*, consists of strong rhythmical oscillations, from about five to seven per

second. It continues in every position, and is usually not increased by voluntary movements; indeed a voluntary movement may stop the tremor for a time. When the tremor is slight, irregularities in handwriting may require a lens for their detection.

The tremor usually begins and is most noticeable in the hands—where it often consists chiefly of the movements of pronation and supination, and may then spread to almost every other part of the body. The trunk muscles, however, are usually, and the abdominal ones invariably, spared. If the tremor is unilateral, and the shaking of the affected hand be forcibly checked, the free hand often begins to shake.

The tremor of **disseminated sclerosis**, sometimes called *paralytic tremor*, is wild and irregular in character. At the commencement of a movement, such as that constituted by taking a glass of water in the hand and carrying it to the mouth, the tremor is slight, but it gradually increases to a violent shaking, so that a straight line is changed into a zigzag one, the oscillations of which show a progressive increase in size, and become wild and irregular. Their violence is frequently proportionate to the effort made to overcome them.

A precisely similar coarse jerky tremor, or series of clonic spasms, as the movements might equally well be designated, occur in some cases of unilateral brain disease, especially perhaps when the lesion is situated in the neighbourhood of the optic thalamus.

The **tremor of old age** is usually of fine quality, and closely resembles that of paralysis agitans, but the following differences may commonly be observed :—

(1.) The head and face muscles are almost invariably attacked, and at an early period, by the tremor of old age; but in paralysis agitans they frequently escape, and when they are affected, it is usually at a much later period than the hand. (2.) Senile tremor is usually bilateral from the first; in paralysis agitans tremor attacks one side of the body before the other. (3.) Senile tremor, when slight, may only appear during voluntary movements, and generally is more influenced by voluntary movements than the tremor of paralysis agitans.

Tremor of intermediate character is, however, frequently observed, and then the diagnosis has to be based on concurrent symptoms, such as the presence of muscular weakness and rigidity in the case of paralysis agitans.

Tremor also occurs in **exophthalmic goitre**, in alcoholic, lead and mercurial **poisoning**; in these cases, as a rule, it is only observed on movement. In exophthalmic goitre sometimes the whole body shakes continuously with a slight gentle tremor; as a rule, the rhythmical oscillations are smaller and quicker than in paralysis agitans or in

senile tremor, and resemble those produced in healthy persons by psychical disturbance, or in various conditions of nervous weakness, as during convalescence from severe disease.

Alcoholic tremor is more marked after fasting, and is temporarily diminished by the use of spirits. A precisely similar tremor to that of "delirium tremens" may be seen in meningitis and in febrile delirium; it must, moreover, be remembered that the condition known as delirium tremens may exist without tremor.

In **hysteria**, while every variety occurs, the tremor is usually of paroxysmal character, and presents jerky transitions between different types, a fine quality, for example, being suddenly replaced by jerky clonic splasm.

Convulsion is the severest form of clonic splasm, and the term is commonly restricted to cases in which most of the muscles of the body are simultaneously affected with clonic spasms. It is convenient, however, to include under this term severe forms of local spasm, such as those excited by lesions in the motor region of the cortex.

In most cases, a convulsion is to be regarded as the result of a discharge of energy from the cells of the cerebral cortex, and hence it indicates an instability of those cells. Such instability may be brought about by many causes:—Thus (1.) By local disease, as meningitis or a cortical tumour. (2.) By blood changes, as in pyrexia; for example, convulsions sometimes occur in childhood at the onset of the acute specific fevers or of pneumonia; also in hypervenous states of the blood, as in congenital heart disease. (3.) Reflexly, as from the presence of a worm or of undigested food in the alimentary canal. (4.) In an unknown way, as in ordinary epilepsy and hysteria.

Clinically, two distinct types of convulsion may be recognised. In the one type the convulsion starts deliberately in a particular group of muscles, then may slowly spread to other muscles, but does not, as a rule, affect the body generally, nor is it attended, except in severe cases, by loss of consciousness. In the other type the convulsion is general, and is commonly attended with loss of consciousness. The former may for convenience be termed local, the latter general convulsions.

Local Convulsions ("**Jacksonian Epilepsy**").—These are caused for the most part by organic disease of the motor areas of the cerebral cortex. When limited to one limb or to one side of the head, they are called monospasms, and the following clinical varieties may be distinguished:—Crural, brachial, facial, oculo-motor and masticatory monospasms. Each of these monospasms is produced by irritation of the particular region of the cortex which governs the movements of the parts involved in the local convulsion, and if the convulsion spreads,

the muscles next to be attacked are those whose motor centres are nearest to that portion of the cortex first irritated. Thus, a convulsion beginning in the leg will spread to the arm before the face; one beginning on one side of the face will extend next to the arm, and attack the leg last of all. In some cases, a convulsion beginning thus locally may eventually become general, and then, as a rule, there is complete loss of consciousness. It is therefore of the greatest importance to observe accurately the initial or "signal" spasm, because that is our guide to the precise situation of the lesion. Thus, in one case in which clonic spasm began in the great toe, a cicatrix was found in front of the

FIG. 214.—Showing Facial Spasm at the commencement of an attack of "Jacksonian Epilepsy." The attacks in this patient always begin at the right angle of the mouth; the head is then rotated to the right side, the tongue is arched and drawn to the right side, and power of articulation is lost; then the upper limb becomes affected, but not the lower limb. The man has been subject to these attacks for several years; consciousness is never lost, and there is not the slightest trace of paralysis in arm or foot. The etiology obscure; no history of syphilis.

highest part of the fissure of Rolando, and when this was removed (by Horsley) paralysis of the great toe only remained. The patient is often able to describe, even during the progress of the convulsion, how and where it began. It may be observed: (1.) That convulsions of this type, while limited to the limbs on one side, may affect muscles which are bilaterally associated in action, such as those of the abdomen and thorax on both sides of the body. (2.) That an attack of local convulsions is usually followed by transitory or permanent paralysis of the affected muscles.

General Convulsions are the most prominent feature of epilepsy;

they occur also in hysteria, and in many cases of organic brain disease, especially when, as in meningitis, the morbid process is a diffused one. We have also just seen that local convulsions dependent on a local cortical lesion may, in severe cases, become general. **Eclampsia** is sometimes used as a designation for the recurrent convulsions which come on in uræmia, in the puerperal state, and in young children apart from brain disease; but the term is not applied to a single fit. Convulsions are more readily excited in young children than in older persons; thus they often occur during the first dentition, especially in rickety infants; are started by the irritation of intestinal worms; and sometimes constitute the initial stage of the acute specific fevers, of pneumonia and of acute poliomyelitis.

A typical *epileptic* fit is made up of three stages—a tonic stage, a clonic stage, and a stage of gradual recovery. The patient, with or without warning, called **aura,**[1] suddenly loses consciousness, then the muscles, first of one side and then of the other side of the body, or less commonly of both sides simultaneously, become quite rigid. In a few seconds the tonic merges into the clonic stage, in which the muscles first attacked with tonic spasm become the seat of clonic spasms; other muscles are quickly attacked, till the whole body is thrown into convulsions. Frequently the tongue is pushed between the teeth and bitten; the saliva is frothed in the mouth; and urine and fæces are passed involuntarily. Towards the end of this stage, which usually lasts two or three minutes, spasmodic jerkings become slower and less frequent, till they finally cease; the patient lying for a time in a state of deep coma with flaccid paralysed limbs; he breathes stertorously, his face is cyanotic, and his pupils are widely dilated. The third stage is characterised by a gradual return to consciousness and voluntary power; during this period of transition, twitching or rigidity of muscles may here and there be noticed; and before complete consciousness is regained the patient may perform various automatic acts of which he has afterwards no recollection.

In rare cases, a grave condition, known as the "status epilepticus," is met with. It is characterised by a rapid succession of fits and by deepening coma. The pulse and respiration are greatly quickened; the temperature may rise to 105° or 107°, and death frequently ensues.

The convulsions of uræmia and of the puerperal state closely resemble those of epilepsy; they set in suddenly, comprise a tonic and a clonic stage, and, as a rule, exhibit a violent character.

Hysterical Convulsions.—In hysteria almost every variety of clonic or tonic spasm, or of general convulsion, may be witnessed. The

[1] This is a term applied to any sensation or motion which is experienced by the patient, and which warns him of the oncoming attack.

convulsive attacks of hysteria can generally be recognised by their intensity, their obstinacy, and by the fact that their onset is usually referable to some unusual emotional excitement. It is also to be observed that the contractions rarely if ever implicate muscles whose movements cannot be voluntarily performed; thus contraction of one half of the frontalis, incoördinate movements of the eyeballs, and clonic spasms of the muscles on one side of the chest or abdomen are never observed in hysteria.

Two types of convulsive attacks have been distinguished under the terms hysteria minor and major.

Hysteria minor is the term applied to the mildest form of convulsion, in which emotional discharges are associated with violent movements of the limbs, arching of the spine, and throwing of the head from side to side.

Hysteria major, *Hystero-epilepsy*, *Hysteroid convulsions* are terms given to the severest and most complex varieties of convulsive phenomena. Three stages are described by French writers.

The first, or epileptic stage, is often preceded by certain prodromata, such as borborygmi, globus, palpitation, &c., or by a period of mental disturbance, with hallucinations; then the patient frequently experiences a sensory aura,—some curious sensation in the hypogastrium or ovarian region, or in the soles of both feet,—which ascends to the neck or head, after which the patient falls down. All the muscles become rigid, respiration is suspended, and the attack closely resembles the first stage of epilepsy. But it will be noticed that the patient is rarely injured by falling; that the hands, though clenched, never exhibit the interosseous position which is often seen in epilepsy; that the tongue is frequently outstretched, that the pupils are normal, and that pressure over the ovarian regions, commonly unduly sensitive, will often arrest the attack.

The second stage, or the "phase des grandes mouvements," is characterised by the most varied and grotesque attitudes. Of these the most striking is the "arc de cercle," in which there is marked opisthotonus, with the head bored back into the pillow; so exaggerated may this posture become, that the trunk is pushed forwards from the bed, and the patient rests on her heels and head, the latter in the severest cases being so thrust back that the vertex, or even the forehead, forms the point of support. Screaming, tearing, biting, and all kinds of struggling movements, are common in this stage, the severity of which is proportional to the efforts made to restrain them. The clonic spasms which occur in this stage are less rapid and shock-like than those of epilepsy, and indeed more closely resemble a coarse tremor than the true epileptic jerkings. Another distinction is that the tongue is

never bitten, although exceptionally both it and the lips may be accidentally bitten as the patient falls to the ground.

The third stage, or the "phase des attitudes passionelles," now appears. During its continuance expression may be given by delirious talk and by attitude to every possible emotion, as anger, fear, joy, grief, or the most intense voluptuousness. Hysteroid attacks may occur in series, like epileptic attacks, constituting what is known as the *status hystericus*. This is distinguished from the *status epilepticus* by the facts that the temperature is not raised and that life is not threatened. Hysteroid convulsions may follow a true epileptic seizure, but in such cases it will usually be found that the patient formerly had epileptic fits which were not followed by such convulsions. They may occur in patients suffering from organic brain disease, the disturbance of nerve function from real disease being the exciting cause of the hysteroid attack. Hence in all seizures apparently hysterical, a careful search should be made for any evidence of organic disease, the symptoms of which may have been masked by the superposed one of hysteria.

The main points of distinction between epileptic and hysteroid fits are brought out in the following table by Gowers:—

	Epileptic.	Hysteroid.
Apparent cause.	None.	Emotion.
Warning.	Any, but especially unilateral or epigastric auræ.	Palpitation, malaise, choking, bilateral foot aura.
Onset.	Always sudden.	Often gradual.
Scream.	At onset.	During course.
Convulsion.	Rigidity, followed by "jerking"; rarely rigidity alone.	Rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting.	Tongue.	Lips, hands, or other people and things.
Micturition.	Frequent.	Never.
Defæcation.	Occasional.	Never.
Talking.	Never.	Frequent.
Duration.	A few minutes.	More than ten minutes, often much longer.
Restraint necessary.	To prevent accident.	To control violence.
Termination.	Spontaneous.	Spontaneous or induced (water, &c.).

Chorea Minor.—This is a condition chiefly met with in children, and characterised by involuntary twitchings or clonic spasms of various muscular groups. In slight cases the twitching movements may be limited to one hand; in severe cases every part of the body is affected, the features undergo every variety of contortion, the tongue is quickly

protruded and quickly retracted and twisted about from side to side, the head and limbs execute every variety of movement. In such severe cases speech is difficult or impossible, and the incessant tossing about produces bruises, and even open sores, on the projecting points of the body and limbs, in spite of every attempt to protect the patient from injury. Between slight and severe cases every variety may be met with; sometimes the irregular contractions involve the muscles on one side of the body only, when the disease is called unilateral chorea or hemichorea. In the severest examples of chorea, signs of endocarditis are almost invariably present; and in slight or moderate examples a mitral murmur is not uncommon. Evidence of rheumatism as articular pain or swellings, subcutaneous nodules, or muscular pains, is also to be looked for.

The movements of chorea usually cease during sleep, and they may be *checked by a voluntary effort*. They are, however, aggravated by excitement, or by withdrawing the patient's attention from the affected parts.

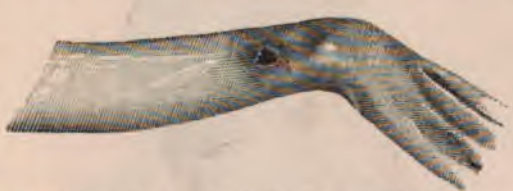


FIG. 215.—Wasting of Extensor Muscles of Right Forearm and of Interossei in a Girl the subject of Chorea following Acute Articular Rheumatism.

Some degree of muscular weakness is usually present, and there may be distinct paralysis, and sometimes muscular atrophy. A chronic progressive form of chorea is sometimes met with in the adult; in one variety, called *Huntingdon's chorea*, a certain amount of insanity is commonly present.

The movements of chorea may be closely imitated by hysterical patients, when the condition is termed hysterical chorea, or chorea major. Sometimes the movements are regular and rhythmical, sometimes they are irregular and varied. But in the latter case they are usually more quick and sudden than in true chorea, and are always *increased by attention* being directed to them.

Various Disorders of Movement are occasionally found in association with the late rigidity of hemiplegia. They more commonly follow the hemiplegia of early than of adult life. At the latter period hemi-anæsthesia is sometimes present, and the lesion is usually in or near the optic thalamus, but in children any part of the cerebral motor path may be damaged. The most frequent form of movement consists of slow irregular muscular contractions; the spasm in the hand affects mainly

the interossei and lumbricales, and thus the posture differs from that of ordinary late rigidity, in which the long flexor of the fingers is contracted. In some cases the movements continue during rest, and then the condition is called *Athetosis* (a term first given by Hammond to cases in which such movements were present without a pre-existing hemiplegia).

In other cases the muscles of the affected side (chest as well as limbs) are affected with a fine tremor; rarely, quick irregular muscular contractions are observed, which, from their resemblance to those of chorea, have been called "post-hemiplegic chorea."

Forced Movements, such as rolling round the longitudinal axis of the body, sometimes result from lesions of the peduncles of the cere-

FIG. 216.—Athetosis in a Young Man the subject of Spastic Hemiplegia, which dates from Infancy.

bellum, and chiefly from damage to the middle peduncle. Vertigo is commonly a marked feature of such cases, and there may be a crossed anæsthesia (loss of sensation of the face on one side, and of the limbs on the other side), with hemiplegic weakness, owing to pressure on or damage to the pons Varolii.

Diminished Muscular Action.—A muscle is said to be paralysed when voluntary power to move it is lost or considerably impaired. Paresis is a term often used to express a minor degree of weakness. In addition to the degree of motor weakness two other points require investigation, namely, (1) the exact distribution of the paralysis, and (2) the condition of the affected muscles with respect to nutrition and tone.

Degree and Distribution of Paralysis.—When motor weakness is marked, there can be no difficulty in defining its limits; whereas the recognition and limitation of slight degrees of paralysis require the greatest care on the part of the investigator. Each movement—flexion, extension, abduction, adduction—and, whenever possible, each muscle, should be separately tested and compared on the two sides of the body; allowance being made for the usually greater strength of the right than the left limbs. When the patient is unconscious, each limb should be lifted up from the body and then allowed to drop, when it will be found, unless the coma be very deep, that the paralysed limb falls down limp and helpless, while the healthy one presents some resistance, and when let go, sinks gradually on to the bed, or is suspended for a time.

For a description of the actions of the various muscles, the student is referred to works on anatomy, but we would draw his attention to the great importance of making a systematic examination of the various movements of the body. It is a common experience in the wards of a hospital to find that the student who has spent some time in examining and drawing up a report of a nervous case is unable to say whether the patient can sit up or turn in bed, or stand erect without assistance. The observation of such simple facts, although quite easy, is frequently neglected, chiefly because the necessity for their investigation is not present in the student's mind at the time of examination.

The omission is avoided by the adoption of a definite plan. Preliminary observations with regard to what may be called the grosser movements of the body should be made before submitting the finer or individual movements to a thorough systematic examination. Can the patient walk, stand, sit up in bed, move all his limbs—are questions which the student must first answer. Then his attention should be directed to that part the strength of which appears to be most markedly impaired, and subsequently to the movements of other parts of the body. As a rule, the strength of a particular movement is best estimated by offering resistance to it; and in this way the various movements of the head, trunk and limbs may be successively investigated.

For example:—(1.) The strength of the hand muscles is ascertained by getting the patient to grasp one's hand; to abduct and adduct the fingers; to touch the tip of his little finger with the end of his thumb (see Figs. 35 and 36, p. 71). A comparison between the grasp of the two hands may also be made by means of the dynamometer, an instrument which also enables us to keep a record of variations in the strength of the grasp at different times. (2.) The power of flexion and extension at the wrist, elbow, ankle, knee, or hip is ascertained by offering

passive resistance to the particular movement, or by forcibly extending or flexing the joint which the patient endeavours to keep flexed or extended. In testing flexion at the elbow, we must remember that the supinator longus is the chief flexor when the forearm is midway between supination and pronation, whereas the biceps mainly acts when the forearm is supinated. (3.) The strength of the deltoid is ascertained by offering passive resistance to abduction of the arm; that of the pectoralis major by getting the patient to try and touch the opposite shoulder, while the observer grasps the patient's wrist and tries to pull it away from the shoulder; that of the latissimus dorsi by offering resistance to what may be called the "the tail-coat-pocket movement," *i.e.*, the patient puts his hand behind him, as if to reach a pocket in a tail-coat, while the physician, who stands behind the patient, grasps the patient's wrist and tries to drag the limb up and out, away from the spinal column.

With similar care, adduction and abduction of the limbs, flexion and extension of the head and trunk, rotation of the head, and

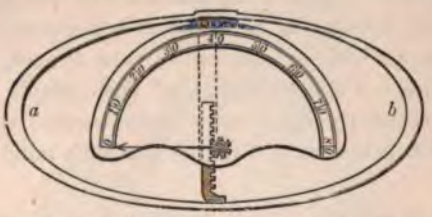


FIG. 217.—Dynamometer of Mathieu. The index registers the force exerted in grasping.

finally, certain special movements, as those of the eyes, tongue and larynx, should be systematically investigated.

Terminology.—Paralysis of one half of the body is called *hemiplegia*; paralysis of one half of the face or of one limb is called *monoplegia*; thus, there may be a monoplegia facialis, brachialis, or cruralis. *Paraplegia inferior* is paralysis of both legs; *paraplegia superior*, paralysis of both arms. When all four extremities are paralysed, the condition, if due to brain disease, is called *diplegia*, *double* or *bilateral hemiplegia*; if due to spinal disease, *paraplegia cervicalis*; *crossed* or *alternate hemiplegia* means paralysis of the limbs on one side, with paralysis of the facial, third, or other cranial nerve on the opposite side of the body.

The **degree** of paralysis supplies information with regard to the intensity of the lesion which causes it, but by itself affords no indications as to its site. Nor can it be said that it is often possible to define accurately the situation of the lesion from a consideration of the **distribution** of paralysis *alone*. It is true that a wide experience of nervous diseases teaches us that paralyses affecting certain parts of the body

depend on lesions situated more constantly in some parts of the motor tract than in others. For example, hemiplegia of the ordinary type is usually due to disease of a certain part of the internal capsule; and a symmetrical localisation of paralysis to the peripheral parts of the limbs —the extensor muscles of the hands and the dorsal flexors of the feet being chiefly affected—is most commonly due to peripheral neuritis. But in other cases this is not so; thus diffuse paralysis of a limb—that is, paralysis which affects all the muscles of a limb in nearly equal degree —may be due to disease of the brain or spinal cord, and more rarely to disease or injury of the nerve plexus which supplies the limb. Perhaps the only instance in which distribution by itself gives certain results is where paralysis affects all the muscles, supplied by a single nerve, and none of those supplied by any other nerve.

Furthermore, it is desirable for the beginner in the study of nervous diseases to start with a method of investigation which will give him a clear and broad grasp of the subject, and which is based on his knowledge of the anatomy and physiology of the nervous system. Such a method is to be found in a consideration of the

Condition of the Paralysed Muscles as regards Nutrition and Tone. —This forms a basis for a clinical classification of cases of paralysis which is at once simple and comprehensive, and is far more important than degree or extent of paralysis for purposes of local diagnosis. It enables us to separate the various forms of paralysis into two great groups. In the one group—**atrophic paralyses**—weakness is combined with wasting of muscular tissue, and the lesion is situated in some portion of the lower segment of the motor tract. In the other group—**spastic paralyses**—weakness is combined with rigidity of muscular tissue, and the lesion is situated in some portion of the upper segment of the motor tract.

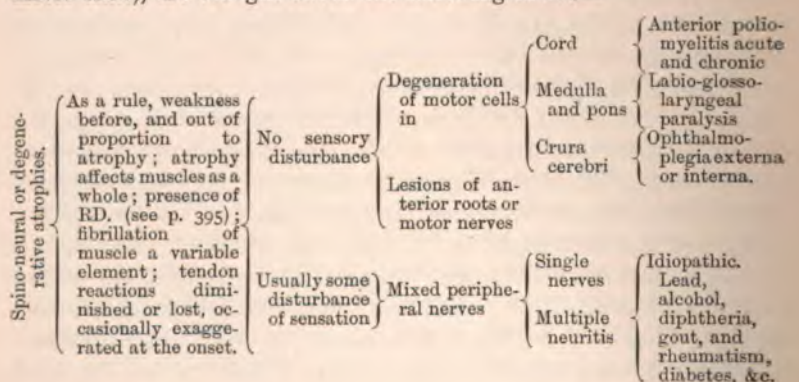
This division does not embrace every form of paralysis, nor is it always possible to determine to which group a given case of paralysis may belong. Thus, (1.) If a limb just stricken with paralysis be examined, the muscles, beyond weakness, may afford no indication as to which variety of paralysis they will ultimately pass into. (2.) In slight degrees of motor weakness, whether of hemiplegic or paraplegic distribution, wasting or rigidity of the muscles may be inconspicuous or absent; in such cases, however, an appeal to the deep reflexes of the affected limb, which are closely connected with the tone of the muscles, may settle the point, for these will usually be exaggerated when the upper segment of the motor path is affected, but diminished or absent when the lesion is situated in the lower segment. (3.) The muscles are merely flaccid, presenting neither rigidity nor wasting in many cases of hysterical paralysis. (4.) It need scarcely be added

that in paralysis of invisible muscles, such as those of the eyeball, it is impossible by observation alone to assert anything with regard to them beyond the degree of their weakness.

Notwithstanding these objections, the plan here adopted (which the author first learned from the late Dr. Ross) constitutes the best working basis, and the student who makes use of it soon acquires considerable facility in diagnosis, at least so far as this relates to the locality of the lesion.

In addition to the two simple groups of paralyses, a third group is constituted by cases in which an atrophic and a spastic paralysis are found combined in the same patient. This may be designated the group of **mixed paralyses.**

Group of Atrophic Paralyses.—The characteristic feature of a well-marked case of atrophic paralysis is the association of muscular atrophy with muscular weakness. Such an association indicates one of two things, either (1) impaired or abolished function of some portion of the lower segment of the motor tract, or (2) disease within the muscle itself. In the former case the atrophy is sometimes termed *degenerative* or *spino-neural;* in the latter *primary myopathic.* But it has yet to be ascertained whether, in the class of so-called primary myopathic atrophies, the disease actually starts in muscular tissue, or whether the muscular atrophy is not really due to disease of the terminal branches of a nerve or of its motor end-plates. If the latter were the case, then all cases of atrophic paralysis would strictly depend on affections of the lower segment of the motor tract, and from a pathological point of view could be classed together. But from a clinical standpoint there is a vast difference between the degenerative and the myopathic atrophies. The more important distinctions between them, as well as the leading points characterising disease of nerve nuclei and of the nerves themselves (the two parts of the lower segment of the motor tract), are brought out in the following table:—



Primary myopathic atrophies:

- Weakness in proportion to atrophy; atrophy invades muscle bit by bit; no RD.; fibrillation absent or present in slight degree; tendon reactions normal, diminished or lost, never exaggerated
  - Atrophy associated with increase in bulk of muscles — Pseudo-hypertrophic paralysis.
  - Enlargement of muscles may be present but oftener absent — Idiopathic progressive muscular-atrophy.

Mode of Investigating a Case of Atrophic Paralysis.—In examining a part affected with weakness and wasting of muscular tissue, the following points require investigation :—

1. The distribution of the weakness or wasting.
2. The proportion that the atrophy of a muscle bears to its weakness.
3. The presence or absence of sensory disturbance.
4. The electrical reactions of the muscles.
5. The condition of the reflexes (see pp. 418–425).
6. The presence or absence of fibrillary tremors (see p. 371).

FIG. 218.—Marked Atrophy of the Small Muscles of the Hand of a Boy, the subject of Disseminated Sclerosis.

I. **Distribution.**—Atrophic paralysis may be limited in extent, or widespread over the muscles of the body.

Limited Paralysis.—When weakness and atrophy affect a single muscle, or a group of muscles supplied by one nerve, the lesion is situated in the motor fibres of this nerve, or in its nucleus of origin.

Examples:—Paralysis of the deltoid from disease of the circumflex nerve; paralysis of the serratus magnus from disease of the posterior thoracic; paralysis of the facial muscles from disease of the facial nerve or its nucleus; paralysis of the extensor muscles of the forearm from disease of the posterior interosseus branch of the musculo-spiral nerve, as in the ordinary form of lead paralysis.

When a group of muscles which act in functional association, but yet are supplied by different nerves, becomes paralysed, there is a lesion of a particular group of cells in the anterior horns, or of certain of the anterior roots, or possibly of the nerve plexus which supplies the affected limb.

Examples:—Paralysis of the deltoid, biceps, brachialis anticus and supinator longus from disease of the fifth cervical root, or of the upper portion of the cervical enlargement of the spinal cord; paralysis of the small muscles of the hand from disease of the first dorsal root, or of the lowest portion of the cervical enlargement (see Fig. 218); paralysis of the peronei and tibialis anticus muscles from disease of the anterior horn, as in infantile paralysis.

Limited atrophic paralysis may have a symmetrical or an irregular distribution. Most of the above examples of paralysis are asymmetrical. A random distribution of weakness and wasting also occurs in

FIG. 219.—Double Wrist Drop, from a Case of Alcoholic Neuritis.

diffuse forms of chronic myelitis. An example of symmetry is afforded by the double wrist drop of lead palsy affecting the extensor muscles of both forearms.

Widespread Paralysis.—This may be due to a chronic degeneration of the anterior horns or to a multiple neuritis. In both cases a symmetrical distribution of the paralysis may be observed, although symmetry is far less common in disease of the cord than in disease of the nerves. The diagnosis, however, is occasionally very difficult. Thus a patient may have double wrist drop and marked atrophy of the thenar and hypothenar eminences, as a result either of peripheral neuritis or of chronic anterior poliomyelitis. In the former case, as a rule, both arms are simultaneously and symmetrically affected; but in the latter the paralysis is commonly unilateral in onset, and even at a late period of the affection is more marked on one side than the other; further, there are usually signs of muscular irritability in the

lower limbs, either spasm of the muscles or exaggeration of the knee-jerks—indications that the fibres of the pyramidal tracts are also implicated. Moreover, the diagnosis is usually cleared up in process of time, for a multiple neuritis may be curable, a chronic anterior poliomyelitis is incurable.

A widespread, or even a universal, paralysis sometimes occurs at the onset of acute anterior poliomyelitis, but then certain parts quickly begin to recover, while others run on to permanent paralysis and atrophy.

The wasting in the myopathic group is usually widespread, but is

FIG. 220.—Photograph of Woman the Subject of Idiopathic Muscular Atrophy, showing Marked Atrophy of Trapezii, and Projection of Scapulæ owing to Atrophy of Serrati.

distinguished from the preceding affections by striking peculiarities in regard to distribution. In well-marked examples, the upper arm, the shoulder, and the thigh are the seats of election; occasionally the face is affected. The thinness of the arm contrasts strongly with the normal-sized forearm, the muscles of which are usually spared with one exception, namely, the supinator longus; this muscle is commonly much atrophied. The lower portions of the pectoralis major and the latissimus dorsi, the serratus magnus, and the lower portion of the

trapezius are also frequently involved. The hands nearly always escape, but both they and sometimes the shoulders are attacked in chronic anterior poliomyelitis, while the face is spared.

Furthermore, it is to be noticed in the myopathic group that the atrophy may be found in association with **enlargement and increased firmness** of certain muscles. Two classes of cases may be distinguished. In the one class, pseudo-hypertrophic paralysis, enlargement of muscles is a conspicuous feature. The muscles of the calf and the infra spinati are most frequently affected. In this disease, then, certain

FIG. 221.—The same Case as Fig. 220, showing wasting of Pectorales, and the drawing up of the Upper Angles of the Scapulæ (owing to the absence of opposition) by the Levator Anguli Scapulæ and upper portion of the Trapezius.

muscles are wasted, others are abnormally large, but whether large or small, the diseased muscles are weak. In the other class, idiopathic muscular atrophy, wasting of muscles is conspicuous, while enlargement of muscles is said to be absent, or, if present, is not a striking feature of the disease.

But it is difficult to make an absolute distinction between the two classes on these grounds, for if cases of idiopathic muscular atrophy be carefully examined, enlargement and hardness of certain muscles, or

parts of them, will often be discovered. Thus, it is common to find the upper portion of the deltoid atrophied, while the lower portion is firm and hard, and to find firmness of some of the forearm muscles in association with atrophy of the supinator longus.

Arthritic Muscular Atrophy.—The atrophic weakness which follows injury or disease of a joint is usually limited to the muscles that move the joint, and is most prominent in the extensor muscles. Thus, if the shoulder is affected, the deltoid chiefly wastes; if the hip, the glutei; if the elbow, the extensor muscles of the forearm; if the knee, the

FIG. 222.—Motor Points of the Facial Nerve, and the Facial Muscles supplied by it. (*Eichhorst.*)

muscles in front of the thigh. The interossei muscles are strikingly wasted in rheumatic affections of the finger-joints.

2. **The Relation between Wasting and Weakness of Muscle.**—In acute lesions of the anterior horns or of the peripheral nerves, weakness is always in excess of atrophy, at least during the active period of the disease. Thus, in infantile paralysis certain groups of muscles may be markedly paralysed before their atrophy is apparent, but at a late period of the affection, atrophy of a limb will be just as conspicuous as its paralysis. In chronic disease of the anterior horns,

weakness and wasting proceed side by side. But the cases in which paralysis is directly dependent on, and proportional to, atrophy of muscular tissue are those already mentioned of idiopathic muscular atrophy. Here muscles waste bit by bit, and their atrophy always precedes and produces their weakness.

3. **Sensory Disturbance.**—The presence of decided sensory disturbance, such as pain, hyperæsthesia or anæsthesia, in cases of atrophic paralysis is sufficient evidence that disease is not limited to the anterior horns, to the motor nerve-fibres or to the muscles, but that it also involves sensory nerve-fibres, or some portion of the sensory tract. Local tenderness or swelling of a nerve-trunk, or limitation of sensory

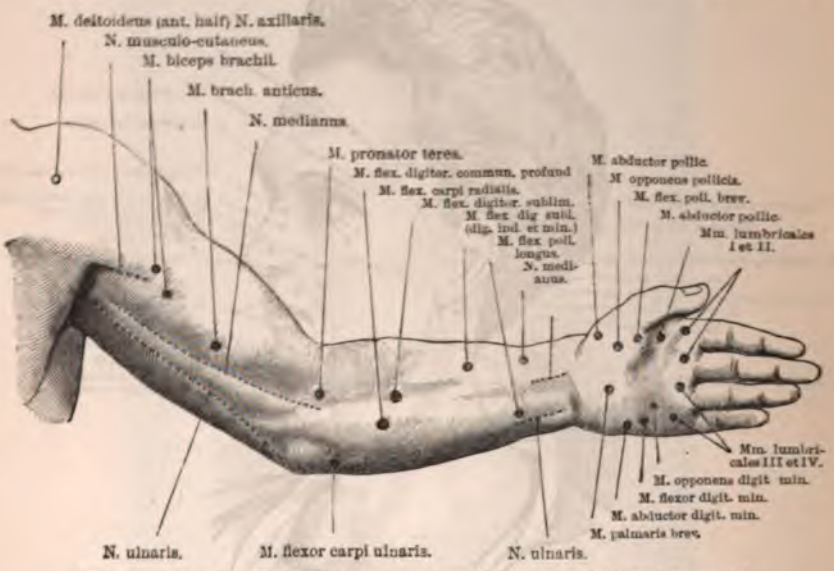


FIG. 223.—Motor Points of the Median and Ulnar Nerves, with the Muscles supplied by them. (*Eichhorst.*)

disturbance and paralysis to the territory supplied by a particular nerve are certain indications of a peripheral nerve affection. Hyperæsthesia of the skin or muscles points to a lesion of peripheral nerves or of the posterior roots. A certain amount of pain and hyperæsthesia of the limbs may be observed in cases where the lesion affects the posterior and anterior horns, and is limited to the cord. There may also be a more or less persistent zone of hyperæsthesia at the upper level of the lesion, but anæsthesia of parts below the lesion is the main sensory phenomenon that is found in association with atrophic paralysis of the muscles when the cord is the seat of disease.

It must, however, be borne in mind that pains like those of rheu-

matism may be present at the onset of cases which turn out to be examples of disease limited to the anterior horns.

4. **Electrical Examination.**—In order to make a satisfactory electrical examination it is necessary to have—1. A faradic battery, such as the sledge induction coil of Du Bois-Reymond, in which the strength of the current (varying inversely with the distance between the primary and secondary coils) may be read on a scale. 2. A galvanic battery provided with (1) apparatus, either a dial collector of cells or a rheostat, by means of which the strength of the current may be altered, and (2) a current reverser or commutator, which is also commonly employed as an interrupter, although it is more con-

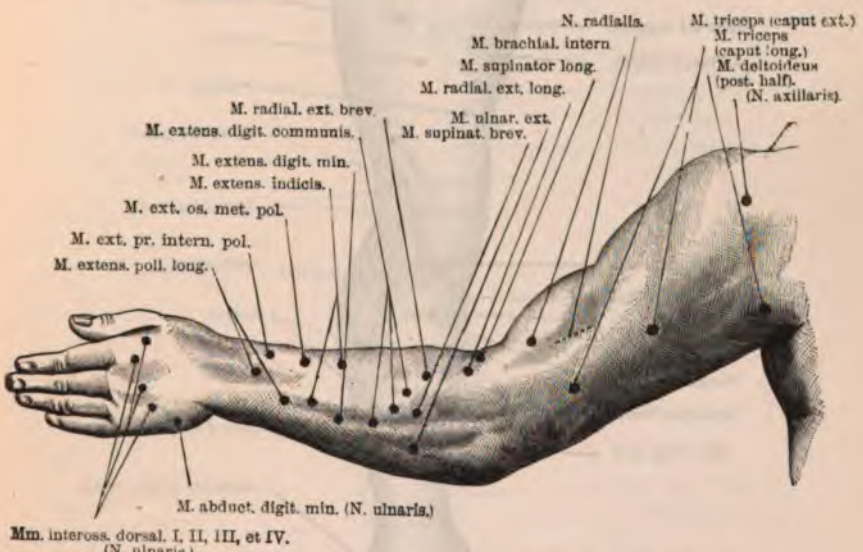


FIG. 224.—Motor Points of the Radial (or Musculo-Spiral) Nerve, and the Muscles supplied by it. (*Eichhorst.*)

venient to have the interrupter in the handle of the electrode; (3) a galvanometer graduated in milliampères; (4) conducting cords or rheophores; and (5) electrodes of various sizes.

Method of Examination.—The patient should be placed in a good light, and in such a position that symmetrical parts to be tested may be equally accessible to the operator. Both electrodes, as well as the skin to which they are applied, must be thoroughly moistened with a solution of salt in hot water. One electrode of large size should be fixed over the upper part of the sternum, the other, of smaller size, is held in the operator's hand, and is successively applied (1) over the trunk of a motor nerve, stimulation of which will cause contraction of

all the muscles it supplies, and (2) directly over the individual muscles. To act on the muscle itself the electrode may be placed either over the point where the motor nerve-branch enters the muscle—the so-called "motor point"—or over some other part of the muscle;[1] in the former case the whole muscle will contract, in the latter only the portion irri-

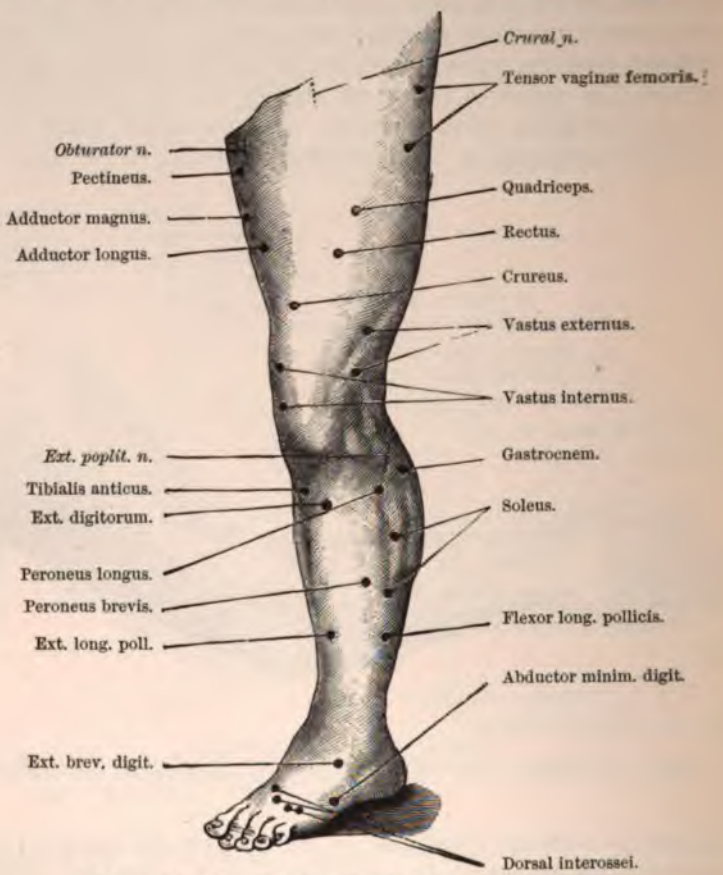


FIG. 225.—Motor Points of Front and Outer Side of Lower Limb.

tated. The position of the *motor points* and the superficial position of the chief motor nerves are indicated in Figs. 222–226 and in Fig. 233.

It is best to begin the examination with the faradic or induced current, and then take the reactions at each pole of the galvanic (called also voltaic or constant) battery; in each case a feeble current should

[1] In health, identical effects are obtained by applying the electrodes over the substance of the muscle and over the motor point, but this is not the case in many diseased conditions.

be first employed, and then gradually increased till a slight contraction is obtained, when the strength of this minimum current, indicated by the deflection of the galvanometer, should be recorded. Sometimes considerable difficulty is experienced in obtaining a reaction, probably owing to an unusual degree of resistance to conduction in the epidermis; it is then advantageous, after thoroughly moistening the skin, to pass a moderately strong current through the part. We are then more likely to obtain accurate results when the muscles are tested with

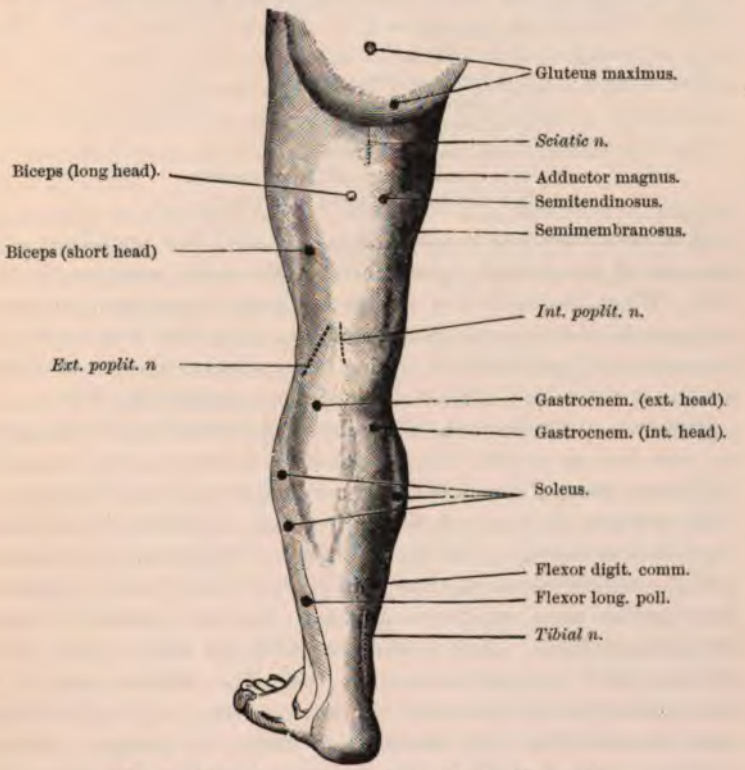


FIG. 226.—Motor Points of Back of Lower Limb. (*Eichhorst.*)

feebler currents. In testing with the galvanic battery, the kathodal or negative electrode should first be placed over the nerve or muscle to be examined, a few cells put in circuit, and the current gradually made stronger, till a slight contraction occurs on its closure. With each addition to the strength of the current the "make" or closure should be tried three times with the commutator or hand interrupter electrode; this is called the kathodal closure contraction (KSC). Then, without removing the electrode, the current is reversed by the commutator; the

exciting electrode is now the positive pole, and in the same way the minimal closure contraction (ASC) is recorded in milliampères by the galvanometer; finally, the contraction at the opening or "breaking" of the current at the anodal pole (AOC), and, if possible, also the kathodal opening contraction (KOC) should be recorded.

Law of normal contraction:—

a. Weak currents produce KSc.
b. Medium ,, ,, KSC′, ASc, AOc.
c. Strong ,, ,, KSTe, AOC′, ASC, KOC.

A = anode.
K = kathode.
c = feeble contraction.
C = medium contraction.
C′ = strong contraction.
Te = tetanic contraction.
O = opening or breaking of current.
S = closure or making of current.

It is seen that ASc is obtained before AOC, but sometimes, as in the case of the musculo-spiral nerve, AOC occurs more readily than ASc. When the belly of a muscle is directly stimulated, we usually only get the closure contractions, and, as a rule, KSc before ASc; but not very rarely, especially in testing large muscles, like the quadriceps femoris or the deltoid, ASc is obtained more readily than KSc.

Pathological Alterations.—The electrical excitability of the nerves and muscles may exhibit changes either as regards quantity or quality, the former being of much less diagnostic importance than the latter.

Quantitative Changes.—A simple increase or decrease in the excitability is determined, in the case of unilateral affections, by comparison with the healthy side of the body; in bilateral affections by comparison with healthy parts which are known to react to currents of nearly the same strength. This is the case with the ulnar, facial, spinal accessory and peroneal nerves; so that if in disease, *e.g.*, of the muscles supplied by the ulnar nerves, a weaker or stronger current were required than that necessary to excite the peroneal (external popliteal) nerve, it would be fair to assume that the ulnar nerves presented quantitative changes. But in coming to such a conclusion the utmost caution is necessary, owing to the various degrees of resistance, not only of different parts of the surface, but also of the same parts in different individuals. It is, therefore, always essential to appeal to the galvanometer, to repeat the observations at least three times, and to ignore differences that are but slight between the healthy and diseased parts. If a quantitative change is present, it points to an abnormal condition of the motor path, and is of value in discriminating between organic and functional affections.

A simple increase has been observed in tetany, KSC being readily obtained by a weak galvanic current. It has also occasionally been observed in hemiplegia, in tabes, and in the early stage of peripheral neuritis.

A Simple Diminution of electric excitability is met with in cases of simple muscular atrophy, as in pseudo-hypertrophic paralysis, and in the other so-called myopathic atrophies, also in the muscular atrophy associated with joint affections and with chorea; also in many of the muscles in cases of amyotrophic lateral sclerosis, and of chronic anterior poliomyelitis, while other muscles give the reaction of degeneration (RD.).

In high degrees of lessened excitability it may be necessary, in order to obtain a muscular contraction, to quickly reverse a strong voltaic current from positive to negative ("voltaic alternation"). If with the strongest current no contraction results, then the galvanic irritability is said to be lost.

Quantitative and Qualitative Changes.—The Reaction of Degeneration.—*Complete RD.*—Unlike the simple quantitative alterations, the nerve and muscle reactions do not run a parallel course, and must therefore be considered separately.

Nerves.—If a peripheral motor nerve is injured or diseased, or in any way cut off from its trophic centre, or if the trophic centre itself is more or less destroyed, motor paralysis is produced, the nerve degenerates, and the degenerative atrophy spreads to the muscles supplied by the nerve.

Sometimes for one to two days after the lesion the nerve excitability increases, but then always begins to diminish, and equally to both currents, and the rapidity of diminution varies with the severity of the lesion. In a case of moderate severity the nerve loses its irritability to both faradism and galvanism by the end of the second week, and the loss continues till about the end of the second month, when slight reactions to strong currents begin to appear, and the irritability gradually increases, but remains for a long time below normal, even after the complete restoration of voluntary power. In an incurable case the non-excitability is persistent.

The Muscles behave in a similar way to the faradic current, although the return of faradic irritability is somewhat slower and more gradual. They differ, however, widely from the nerves in their behaviour to galvanism. The galvanic irritability sinks at first, but during the second week begins to rise above normal, and sometimes to increase during the third and fourth weeks, when muscular twitchings may be excited by only a few cells. Instead, however, of the lightning-like contraction seen in healthy muscles, the movement is slow, delayed and prolonged, and if the current is continued is apt to become tetanic.

Also ASC increases till it equals or surpasses in intensity KSC, and opening contractions may also be obtained, when KOC may be equal to or greater than AOC.

The galvanic muscular irritability continues above normal for one to two months, then sinks, while the qualitative changes remain. In incurable cases the galvanic irritability falls lower and lower, till finally a weak sluggish response to ASC is the only evidence that muscular tissue still exists, and in a year or two even this slight reaction may vanish.

Partial RD.—Here nerve excitability to both currents is preserved, and sometimes the muscles act normally to faradism; but they present the same changes to galvanism as in the complete RD.

Between the complete and the "partial" type of the RD here described, and between the latter and healthy reactions, there are many varieties, and it is often difficult, when the departure from normal is not great, to say whether or not degenerative atrophy has commenced.

The increased excitability of the muscles to galvanism, perhaps only of short duration, may have disappeared before the patient comes for examination, or the undue response at the positive pole may not be distinct, especially in the later stages; it may indeed be found that KSC is equal to or greater than ASC. There is one sign, however, that is never wanting—namely, sluggishness of muscular contraction. If the movement is distinctly slower and less lively than in health, the presence of some degree of degenerative atrophy may safely be inferred. The RD occurs in peripheral lesions of the motor nerves, whether of traumatic, rheumatic, alcoholic, or diphtheritic origin; and in diseases affecting the anterior horns of the spinal cord and the nuclei of the medulla oblongata, as in infantile paralysis, diffuse myelitis, bulbar paralysis and amyotrophic lateral sclerosis. In some cases of lead poisoning the RD has been proved to exist in muscles which appeared to have complete voluntary power.

It is of importance to remember (1) that there is no necessary relation between paralysis and the reaction of degeneration, and (2) that no direct information as to the *nature* of the lesion—whether inflammatory, toxic, or traumatic—is afforded by an electrical exploration of the nerves and muscles.

Group of Spastic Paralyses.—In this variety of paralysis the muscles maintain their normal bulk, but their tonicity is increased and the affected limbs present varying degrees of rigidity. The increase of tonus may be so slight that it only shows itself by a very slight increase of resistance to passive movements, or so great that the muscles feel as hard as boards even when the limb is at rest, and render movements by their antagonists totally impossible.

Another peculiarity of spastic paralysis is that the paralysis is diffuse, that is, all the muscles of a limb or of a portion of a limb are paralysed, and although some muscles are weaker than others, as, for example, the extensors than the flexors, it is rare for any muscle situated within the affected territory to escape some degree of paralysis. In this respect it differs from the atrophic paralyses, which for the most part pick out particular muscles or groups of muscles.

Spastic paralysis is evidence that some part of the upper segment of the motor path has its functions impaired or abolished. The condition of the affected part will be the same whether the lesion be situated in the motor cells of the cortex, in the pyramidal fibres, or in their terminal ramifications in the grey matter of the spinal cord.

The Localisation of the Lesion in a case of spastic paralysis is mainly determined by a consideration of the distribution of paralysis, together with the presence or absence of sensory and other associated symptoms.

Spastic paralysis may be hemiplegic, monoplegic, or paraplegic in distribution.

Hemiplegia.—In the ordinary type some muscles on the affected side are completely paralysed, whilst others are merely weakened or remain entirely unaffected. Thus the limbs and the lower half of the face are paralysed, and the tongue, when protruded, deviates towards the paralysed side; but the muscles of the upper part of the face, those of mastication and the muscles of the trunk, present varying degrees of immunity. And, speaking broadly, the degree of paralysis of a part varies directly with the degree of its unilateral use; for example, the arm, which is habitually used without its fellow, is more paralysed than the leg, while the muscles of the eyes, neck and trunk, which are nearly always bilaterally combined, escape. Weakness of the latter may, however, often be detected when the patient makes a strong effort; thus, during a deep inspiration, the affected side of the thorax will be found to expand to a less extent than the healthy side; and when the patient tries to keep his eyes tightly closed, the observer will often be able to detect a diminished resistance of the orbicularis palpebrarum on the hemiplegic side. It should also be noticed that *an emotion* may cause a muscle to contract when a voluntary effort is unable to do so; for example, the paralysis of the lower facial muscles may pass unnoticed when the patient smiles, but is at once apparent when he tries to show his teeth.

After an attack of hemiplegia the paralysed limbs are affected with tonic spasms, which, according to the time at which they come on, are named as follows:—There may be an "*initial rigidity*," lasting for a few hours; then an "*early rigidity*" of a few weeks' duration, to be succeeded by a "*late rigidity*," which persists as long as the paralysis.

In the upper limb there is usually adduction of the shoulder and pronation of the forearm, with flexion of the elbow, wrist and fingers, especially of the distal phalanges. In the lower limb extension predominates, while the foot usually presents talipes equino-varus. Such late rigidity lessens during sleep, is partly overcome by placing the limb in warm water, or by faradising the antagonistic muscles; prolonged passive extension and friction of the muscles also tend to promote their relaxation. Eventually the active contraction passes into a "*structural rigidity*," which is necessarily irreducible.

The varieties of tonic spasm to which a hemiplegic limb is subject vary very greatly in regard to the degree of rigidity, and sometimes (espe-

FIG. 227.—Extreme Talipes Equino-Varus in a case of Hemiplegia dating from infancy.

cially, perhaps, in cases of syphilitic thrombosis) there is a considerable degree of weakness without any appreciable spasm.

When recovery takes place in hemiplegia, the *least paralysed movements* are the first to regain power, while the specialised movements of the hand and fingers, which suffer most, are the last to recover. Recovery is commonly more complete in children than in adults, *but the affected limbs are often arrested in their growth, and the mind may be defective.*

The above description applies to the ordinary type of hemiplegia, sometimes called "total hemiplegia." This, when persistent, is due to a lesion of the cortex or of any part of the pyramidal tract above the middle of the pons; when transient, to disease in the neighbourhood of the motor path or centres, injuring or compressing them. Total

hemiplegia is rare from disease of the cortex or the centrum ovale, and usually depends on a lesion of the internal capsule, affecting the anterior two-thirds of its posterior segment. Hemianæsthesia and hemianopsia in association with hemiplegia indicate disease of the posterior end of the internal capsule, and then, too, the functions of the other special senses may be impaired on the same side as the hemiplegia: in such cases the leg is more paralysed than the arm, and indeed motor weakness is rarely permanent, and may be only slight in degree. Right-sided hemiplegia is usually associated with some degree of motor aphasia, which, however, soon passes away unless the lesion involve the speech-centre in the cortex, or be situated immediately beneath it. Slight ptosis on the hemiplegic side suggests disease in or near the cortex, probably of the parietal lobe. Paralysis limited to the limbs on one side, the face and tongue escaping, depends in all probability on a lesion of the upper portion of the motor cortex; but in rare cases the lesion involves one of the anterior pyramids of the medulla or the pyramidal tract in the cord. In the latter case the lesion will be on the same side as the paralysis. When a lesion is so situated that one of the motor cranial nerves is involved as well as the motor path, the muscles supplied by the cranial nerves are in a state of atrophic paralysis, whereas those of the limbs are affected with spastic paralysis. Such combinations will be referred to when the mixed paralyses are considered.

Hysterical Hemiplegia differs from hemiplegia of organic origin in the following particulars:—The paralysis is rarely complete in both limbs, and the face and tongue almost always escape; occasionally they are affected by a hemispasm which curls the protruded tongue towards the paralysed side, and then the paralytic deviation of organic hemiplegia is closely imitated. The paralysed limbs may be flaccid, but not infrequently are the seat of contractures, which are characterised by the varying degrees of resistance they present to attempts made to overcome them at different times.

In hemiplegic contraction of some standing, the whole limb cannot be straightened out at once: if, for example, the fingers are straightened, the wrist remains rigidly flexed; but in the hysterical contraction the fingers and wrist may be extended at the same time. The superficial reflexes are often lost on the affected side in organic, but frequently remain normal in hysterical hemiplegia. The latter is also distinguished by its association with hemianæsthesia, impairment of the special senses, and with various emotional disturbances.

Monoplegia.—Some writers apply this term to limited forms of paralysis, whether of spinal or cerebral origin, in which all or almost all the muscles of one side of the face or of a single limb are involved;

and hence they speak of a spinal and of a cerebral monoplegia. But the term is more commonly restricted to partial varieties of hemiplegia, which depend on lesions situated either in or immediately beneath the motor convolutions, so that, according to the seat of the lesion, there may be a crural, a brachial, a facial, a brachio-crural, or a brachio-facial monoplegia. These monoplegias are distinguished by the frequency with which the paralysed parts or some part of the affected side are subject to attacks of partial epilepsy. The clonic spasms, as we have already seen (see p. 374), always begin locally, then may spread to the whole of the side paralysed, and ultimately, in some cases, to the opposite side. As a rule, in crural or brachial monoplegia the paralysis is not absolute; the lowest part of the limb is most affected, and frequently presents some impairment of tactile sensibility or of muscular sense. When the leg alone is paralysed, the lesion is usually situated on the mesial aspect—that is, in the paracentral lobule—or extends but a little on to the outer surface of the hemisphere; for unless disease of the cortex be close to the longitudinal fissure, the arm becomes involved, and then there is a brachio-crural monoplegia. In a facial monoplegia the muscles on one side of the face are affected in the same relative proportion as in ordinary hemiplegia.

As a rule, facial paralysis of cortical origin is complicated by paralysis of the arm, and, when the left hemisphere is the seat of the lesion, by aphasia also. Pseudo-bulbar paralysis, a condition characterised by symptoms closely simulating those of a progressive labio-glosso-laryngeal paralysis, is sometimes produced by bilateral lesions in the lowest portion of the motor region, or by a lesion in the lenticular nucleus of both hemispheres. The general history of such cases is, that the patient has an attack of right hemiplegia, from which he slowly recovers; this is followed in a few weeks by an attack of left hemiplegia and aphasia, the symptoms of which also slowly abate in severity, and then it is found that there is paralysis of the muscles of mastication, of articulation, and to some extent of deglutition.

Hysterical Monoplegia.—Paresis of one limb with marked contractures may follow severe emotional disturbance. It is usually associated with manifestations of the hysterical condition.

Paraplegia.—A spastic paraplegia indicates disease at the top of each motor area in the cortex, or in some portion of the pyramidal tract between these areas and the centres in the lumbar portion of the cord which preside over the muscles of the lower limbs. For the production of a pure example of spastic paraplegia, that is, spastic paralysis of the lower limbs, unattended by any other symptoms, there must be disease affecting both pyramidal tracts, and restricted to them. It is still doubtful whether such a condition, which has been called primary

lateral sclerosis, really exists; certainly in the majority of cases which appear to be of this nature, a careful examination will reveal some impairment of the superficial abdominal reflexes, or other indications that the grey matter of the cord is also implicated. The cases referred to are usually examples of a chronic myelitis, in which the main stress of the disease is on the lateral columns; or a disseminated sclerosis, in which the other symptoms of this disease, such as nystagmus, scanning speech, and tremors on voluntary movement, are not conspicuous, or have not yet developed.

Perhaps the purest examples of spasmodic paraplegia are to be found

FIG. 228.—Case of Congenital Spastic Paraplegia, associated with mental impairment.

in birth-palsies, which occur as a result of meningeal hæmorrhage at birth, leading to atrophy of the central convolutions. Such congenital spastic paraplegia may exist alone, but generally it is found associated with difficulties of articulation, mental defect, or other evidence of cerebral disturbance; it is rare, also, not to be able to detect spasm or other derangement in the movements of the hand.

It is worthy of note that in all cases of spastic paraplegia, whether produced by brain or cord disease, the relation between spasm and paralysis of muscle is very variable. They may be associated together

in almost equal proportion, but frequently it will be found that while a limb may exhibit a fair amount of power, each movement is interrupted by violent spasm, and the tendon reflexes are greatly exaggerated. In other cases slight spasm is associated with considerable weakness.

Hysterical or Functional Paraplegia.—Paralysis of the lower limbs may develop suddenly in consequence of some emotional disturbance, or occur in anæmic subjects apart from definite symptoms of hysteria. The limbs may be flaccid or present marked contractures. In either

Fig. 119.—Spasm of Gastrocnemii and of Adductors of Thighs—the Spastic Paraplegia following convulsions which occurred immediately after birth. (Rose.)

case the knee-jerks are active or exaggerated, there may be ankle clonus, which usually consists of a few jerks slowly evolved, and often somewhat difficult to develop. Sometimes, however, ankle clonus is well marked, and its characters may be indistinguishable from those which constitute the typical ankle clonus of organic disease. The plantar reflex is frequently absent. Commonly there is spinal tenderness, and when the patient can stand she complains of an aching back, and frequently that her legs suddenly give way under her. When a patient suffering from spastic paralysis of organic origin places her back against a wall, and tries to raise the anterior part of the foot

from the ground, spasm of the calf muscles ensues, which fixes the toes still more firmly on the ground; but in hysterical paralysis the patient is able, and may be induced at times, to raise the anterior part of the foot from the ground.

Sometimes in hysteria there is a spastic weakness of all the limbs associated with tremor of more or less changeable type. This condition may present a close similarity to disseminated sclerosis, especially in the early stages of this disease; and when tremor or voluntary movement, nystagmus and scanning speech are absent, the differential diagnosis may be very difficult.

As regards disseminated sclerosis, it is useful to remember (1) that a temporary paresis of one limb may be replaced by a temporary paresis of another limb; (2) that obscurity of vision of one eye may be recovered from, and then be followed by obscure vision in the other eye; (3) that pallor or atrophy of the optic disc is not uncommon. In hysteria, on the other hand, a shifting of powerlessness from one side to the other rarely occurs, and the characteristic affection of the vision is blindness on one side, the optic discs, however, being perfectly normal. (Buzzard.)

Mixed Paralyses.—This heading comprises a large number of cerebral and spinal affections, in which spastic and atrophic paralyses are combined in various proportions, and are found associated with sensory, vaso-motor and nutritive disorders.

In order to unravel the complicated symptoms of such cases, it is essential to have an accurate knowledge of the two simple classes of paralysis just described; to be acquainted with the anatomy of the brain and cord, and to know the functions of the cord at different levels, as well as of its roots and principal nerves.

A few examples will suffice to illustrate the general principles upon which a diagnosis is to be made.

Crossed Paralysis.—When hemiplegia is found in association with paralysis of a cranial nerve on the opposite side of the body, the lesion is in or near the origin of that cranial nerve. Thus simultaneous paralysis of the right limbs and the left third nerve indicates a lesion of the left crus cerebri; right hemiplegia with paralysis of the left fifth, a lesion in the middle of the left side of the pons; right hemiplegia with paralysis of the left sixth or seventh nerve, a lesion in the lower part of the pons; right hemiplegia with paralysis of the left side of the tongue, a lesion on the left side of the medulla.

Diseases of the Cord and its Membranes afford many examples of mixed paralysis. The commonest feature of cord disease is paralysis of the lower limbs, which, as we have seen, is of the atrophic or spastic variety, according as the lesion is in or above the lumbar enlargement.

In the latter case the muscles of the trunk or arms may also be implicated. The various disorders of sensation, of the reflexes, and of the functions of the bladder and rectum which may accompany the paralysis, and which are described in another place, are also of great help in determining the situation and extent of lesion. Thus the upward extent of paralysis and of the impaired sensation, together with the position of any pain or zone of hyperæsthesia around the body, supply important information as to the height of a transverse lesion of the cord, while the condition of the reflexes, the nutrition and tonicity of the muscles, give indications with regard to the vertical downward extent of the lesion.

The following groups are given as illustrations of some of the principal types of spinal disease.

Group I.—Cervical Pachymeningitis with Transverse Myelitis. Here disease of the anterior roots gives rise to an atrophic paralysis in the upper limbs; disease of the posterior roots to sensory disturbance at the level of the lesion, whilst interference with the conducting paths in the cord produces a spastic paraplegia together with anæsthesia of parts supplied below the level of the lesion. The exact height of the lesion may be determined by a knowledge of the functions of the anterior and posterior roots of the cervical nerves (see tables in anatomical introduction). Thus in pachymeningitis on a level with the eighth cervical and first dorsal roots, the flexors of the wrist and the small muscles of the hand are weak and wasted, hence there is a tendency to extension of the wrist, and the fingers are held like claws, there is also a band of anæsthesia along the inner side of the hand, forearm and arm. Whereas anæsthesia along the outer side of the arm and shoulder with paralysis of the deltoid, biceps and supinator longus are indications that the fifth cervical root is involved. The symptoms of amyotrophic lateral sclerosis at certain periods of the disease may resemble those of pachymeningitis of the cervical region. But in the former disease the atrophy of the upper limb muscles is less random in distribution than in the latter, and is associated with muscular tension and contractures; and there is an absence of severe pain, of hyperæsthesia and anæsthesia.

Group II.—Transverse Myelitis in lower part of mid-dorsal region. Spastic paraplegia with anæsthesia of the lower limbs and lower part of the abdomen; weakness with degenerative atrophy of some of the muscles of the lower part of back and abdomen. Ankle clonus and exaggerated knee-jerks. Disturbance of the vesical, rectal and sexual functions. Epigastric reflex present, but abdominal and cremastric reflexes absent. Plantar reflex exaggerated.

Group III.—Transverse Myelitis implicating middle and lower por-

tions of the lumbar enlargement. Atrophic paralysis affecting to varying degree the majority of the muscles in lower limbs. Anæsthesia in territory of nerves supplied by sacral plexus, and in portions of territory supplied by lumbar plexus—knee-jerks lost. The plantar reflexes also abolished, but the cremasteric present. Paralysis of the bladder and rectal sphincters, with impaired sexual power.

Group IV.—Disseminated Myelitis: one focus of inflammation in right portion of cervical enlargement; another in left side of lumbar enlargement. Atrophic paralysis of some groups of muscles in right arm and in left leg; spastic paralysis of right leg—anæsthesia of certain parts of left arm and right leg—other symptoms according to number and position of other inflammatory foci in the cord.

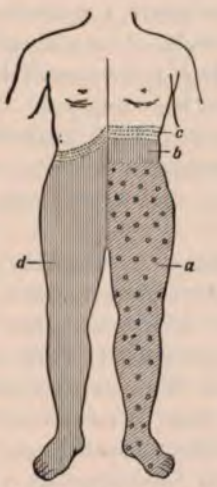


FIG. 230.—Diagrammatic representation of a Lesion of the Left Half of the Spinal Cord in the Dorsal Region. *a*, oblique lines—motor and vaso-motor paralysis; *b*, *d*, vertical lines—complete anæsthesia; *c*, hyperæsthesia, which affects also the left lower limb, as represented by dots. (*Erb.*)

Group V.—Unilateral Lesion of the Cord; the left half of the mid-dorsal region being destroyed. Anæsthesia of the right leg and right half of the abdomen nearly up to the level of the lesion, where there is a band of hyperæsthesia. Paralysis of the left leg, the skin of which is hyperæsthetic; above the hyperæsthetic area of skin there is usually an anæsthetic zone, which corresponds with the level and vertical extent of the cord lesion; above this zone a narrow hyperæsthetic belt may sometimes be detected, which joins that on the opposite side (see Fig. 230). Loss of muscular sense in the paralysed leg; also vaso-motor disturbance on the side of the lesion, usually declared by an elevation of temperature of the paralysed limb.

The reflex actions on the side of the lesion are usually increased, but are not materially altered on the other side.

Paralysis of the Bladder and Rectum.

The wall of each viscus contains muscular fibres to expel the contents, while the presence of a sphincter at the mouth of each prevents continuous evacuation. These muscles are under the control of centres in the lumbar enlargement of the spinal cord, which are themselves partially governed and brought under voluntary control by means of fibres passing between them and centres in the cerebral cortex. The lumbar centre is connected with the periphery by means of afferent and efferent fibres, the former passing from the mucous membrane lining the viscus to the lumbar centre, the efferent from this centre to the sphincters and muscular walls of the viscus.

Complete and permanent relaxation of the sphincters only occurs when the lumbar centres are destroyed. Thus, in cases of myelitis implicating the lower portion of the lumbar enlargement, there is frequently "**paralytic incontinence**" of urine and fæces—that is, urine dribbles away continuously, and fæces escape as soon as they enter the rectum.

Intermittent incontinence of urine may occur when the voluntary path above the lumbar centre is interrupted. Then any sudden movement or pressure on the bladder will cause the urine to be expelled. But loss of voluntary power leads to weakness of the detrusor muscle; hence the bladder is never thoroughly emptied, and when the detrusor is completely paralysed there is retention of urine. Associated with retention there is "**overflow incontinence**," because there is enough pressure in the bladder to force urine out when the sphincter relaxes. Overflow incontinence is a more serious condition than paralytic incontinence of urine, because it indicates incomplete emptying of the bladder, the too frequent results of which are decomposition of urine, cystitis and serious kidney disease.

Retention of urine and overflow incontinence occur not only when the voluntary path is interrupted—as, for example, by a myelitis in the dorsal portion of the cord—but also from disease of the cerebral cortex, especially when this entails lowering of the mental functions. Imperfect emptying of the bladder, and a tendency to retention and incontinence of urine, are met with in locomotor ataxia.

As Dr. Gowers has pointed out, the condition of the sphincter ani is best tested by introducing the finger into the rectum. When the lumbar centre is destroyed, the finger feels a momentary contraction, due to local stimulation of the sphincter, but this is followed by com-

plete and permanent relaxation. But when the centre is intact, and disease is situated at a higher level—as, for example, in cases of transverse myelitis of the dorsal cord—then the initial relaxation of the sphincter which follows introduction of the finger is succeeded by firm tonic contraction.

Perversion of Muscular Action, there being no necessary alteration in Strength—Incoördination of Movement—Ataxia.—In every voluntary movement several muscles are brought into play, and if the movement is to be successful, each muscle used must contract in such a way and to such a degree that its strength is accurately proportioned to that of its fellows. Thus, when the hand is stretched out, and a small object, such as a pin, picked up from the table, the contractions of the many muscles employed, from those of the scapula to those of the finger and thumb, must be exceedingly accurately adjusted, for if there is the slightest irregularity in the balance, either of individual or of associated activity, then the object is missed. The power of selecting the right muscles and of correctly regulating their activities is called **coördination**; and a movement executed in the manner prescribed is called a **coördinated movement**. But when there are errors in the balance or equilibrium of the contractions of the different muscles then the movement is **incoördinated**, and the condition is called **incoördination** or **ataxia**.

Accurate adjustment of muscular action is necessarily impaired by weakness, or by spasm of any muscle employed in a particular movement; but such imperfections of movement are not included under the term "ataxia." And although paralysis or spasm may be found in association with ataxia, the latter frequently exists without either. But while it is true that a want of proportion may be found between the actions of individual muscles, apart from detectable alteration in their strength, it is nevertheless sometimes difficult to discriminate between cases of ataxia and cases where certain muscles are weak or the seat of spasm. For example, in many cases of writer's cramp there is spasm, and sometimes weakness of the special muscles used in writing, but in other cases there is no obvious spasm or weakness, and it may be difficult to give a mechanical explanation of the muscular irregularities displayed in the attempt to write, such muscular irregularities often closely resembling the disorderly movements of ataxia. The difficulty referred to, however, is mainly limited to minor defects in muscular action; for, as a rule, a careful examination will enable us to decide whether certain defects in the movement of a part are due to incoördination or to paralysis. Also, when the two conditions are combined, as in ataxic paraplegia, the incoördination of movement is

still recognisable unless paralysis is profound. Similar remarks might be made with regard to the motor defects met with in multiple peripheral neuritis, especially when caused by alcohol. The gait in alcoholic paralysis has indeed frequently been called ataxic, but in the majority of cases its peculiarities are clearly due to weakness of certain groups of muscles; in rare cases, however, of peripheral neuritis true ataxia does occur.

The actions of a child learning to walk have sometimes been quoted as an illustration of incoördination occurring in health. But the peculiarity of the gait of a young child is the want of dorsal flexion of the foot, and the consequent marked flexion of the thigh on the body in order to clear the foot from the ground. This is imperfect voluntary control over the flexors of the foot on the leg, and not ataxia.

Incoördination of movement is most conspicuous in tabes dorsalis or locomotor ataxia. In this disease it is usually earliest and most strikingly seen in the lower limbs. If a patient, while lying on his back, be told to touch the knee of one leg with the toe of the other foot, or to describe a circle in the air with his foot, the ineffectual efforts of the disorderly movements of the limbs are well brought out. The incoördination is always increased by closure of the eyes (*Romberg's symptom*). Thus at an early period of the disease the patient, when standing with his feet close together and his eyes shut, totters and sways, and may even fall to the ground. Curiously enough, closure of the eyes often increases the unsteadiness of an ataxic patient who is *quite blind*. Romberg's symptom is most marked when sensation is lost in the soles of the feet, but it *also occurs when the cutaneous sensibility is quite normal*. It must also be observed that great muscular power, as exhibited in resistance to passive movements, may be associated with the most marked ataxia.

When tabes dorsalis is fully developed, the patient can only stand securely, even with his eyes open, when the feet are wide apart, and the great effort to maintain equilibrium is evinced by the irregular movements of the tendons on the backs of the feet. The disorderly movements of the legs in walking also testify to the presence of incoördination (see ataxic gait, p. 78).

In Friedreich's disease the upper limbs may be affected at the same time as the lower, but in ordinary tabes their movements become disorderly at a later period of the disease. At first there is inability to execute delicate movements, such as writing; the patient is also unable, when his eyes are closed, to touch a prescribed spot, such as the tip of his nose: when the ataxia of the hand is well marked, the fingers fumble in buttoning and unbuttoning the clothes, an object is reached and grasped in a roundabout and uncertain fashion; and at

an advanced stage of the disease the patient may be totally unable to dress himself or to convey food to his mouth.

Ataxia may occasionally affect the muscles of the trunk. Thus, Dr. Gowers mentions a case of locomotor ataxia in which the patient "could sit steadily on a chair when his eyes were open, but if he closed them would at once fall off."

In severe ataxia involuntary movements, sometimes indistinguishable from tremor, may affect an outstretched limb. This has been called **static ataxia.**

Incoördination of movement, closely resembling that of locomotor ataxia, occasionally occurs in diphtheria and as a sequela to the acute

FIG. 231.—Two Brothers presenting the characteristic Symptoms of Friedreich's Disease, viz.:—Incoördination of movement, absence of knee jerks, jerky tremor, lateral nystagmus, slurred hesitating speech, and curvature of the spine. (*Brain*, 1886.) For a description of the foot deformity and the gait, see pp. 74, 79.

specific fevers. It may also be observed in some cases of hysteria; the patient may be unable to stand steadily when the eyes are closed, and all voluntary movements may exhibit a jerky unsteadiness.

Ataxic Paraplegia.—In this disease ataxia is combined with a spastic weakness of the lower limbs. The knee-jerk is exaggerated, not lost, as in locomotor ataxia, and sensory disturbances are rarely present.

Reeling Movements, in which the patient sways from side to side or from front to back, occur in cerebellar disease, and chiefly when the middle lobe is involved. The gait in such cases is very like that due to alcoholic intoxication. The unsteadiness occasionally resembles that of locomotor ataxia, but, as a rule, the irregular jerky movements of

the legs are not present; the staggering is due rather to a swaying of the whole body than to disorderly movements of the lower limbs. This "cerebellar ataxia" rarely involves the upper extremities. It is often associated with vertigo, severe headache, and optic neuritis, and there may be indications of motor paralysis from pressure on the pons or medulla. Similar derangements of coördination occur in Menière's disease, in combination with vertigo, noises in the ears and deafness on one side. Sometimes there is vomiting, but in uncomplicated cases marked headache, optic neuritis and paralysis of the limbs are absent.

DISORDERS OF SENSATION.

Disorders of sensation may result from injury or disease of any portion of the sensory apparatus, whether it be a peripheral end organ, as the eye, or a tactile corpuscle; a sensory nerve-fibre or a ganglionic nerve-centre.

They comprise: (1.) An excess of the normal sensibility of a part, *hyperæsthesia*. (2.) A diminution or loss of the normal sensibility of a part, *anæsthesia*. The terms *hyperalgesia* and *analgesia* are sometimes employed when excess or diminution of sensibility is limited to the application of painful stimuli. (3.) The presence of *pain* in the absence of an external stimulus. (4.) The presence of abnormal sensations, such as tingling, numbness, crawling, itching, "pins and needles," feelings of cold or heat. These may occur apart from any external stimulation, and are called *paræsthesiæ*. (5.) Acceleration and retardation of sensory perception.

In the present section attention will be given to the chief disorders of cutaneous and muscular sensations.

The examination of the cutaneous sensibility is often attended with difficulty, and requires much patience on the part of the investigator. He has to rely on the intelligence, honesty and goodwill of the patient, and must be constantly on his guard against erroneous statements, whether intentional or unintentional. The patient may complain of numbness in certain parts, which, when examined, appear to be normally sensitive, or he may be totally unaware that the sensibility of a part is increased or diminished until it is objectively tested. Again, when the loss is only partial, the boundaries of the affected part may vary with each examination, and it may require much skill and judgment to strike an average.

In applying the tests, the following rules should be observed:—

1. The patient's eyes must be closed.

2. Direct him to say "Yes" immediately he feels the skin touched, and let him then indicate the part touched.

3. Compare corresponding points on the two sides of the body.

Tactile Sensibility may be tested by means of the observer's finger, or by a light touch with a feather.

The Sense of Locality is tested by asking the patient to indicate with closed eyes the part touched, and in health the error is small. A more accurate test is to be found in the minimal distance at which two points touching the skin are recognised as two. This test may be applied by means of a pair of compasses, or by an æsthesiometer, such as that devised by Sieveking. The distance at which the points are recognised as separate varies in health according to the part of the body touched, being, for example, 1.5 mm. at the tip of the tongue, 2.3 mm. at the tips of the fingers, and as much as 70 mm. on the upper arms and thighs.

The power of discrimination varies, too, with the intelligence of the patient, and may be increased by practice.

The Sense of Pressure is tested by applying different weights to the

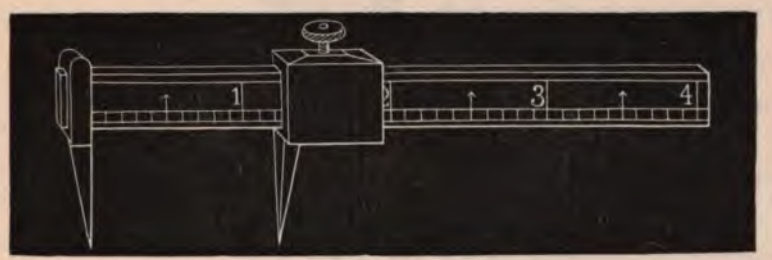


FIG. 232.—Æsthesiometer of Sieveking.

parts to be examined, which should be properly supported, in order to exclude the sense of muscular contraction. Also, it is desirable to interpose a disc of wood, or other non-conducting substance, to exclude the sense of temperature. The maximum variation recognisable in health is about one-twentieth of the total pressure.

Sensibility to Pain is best tested by pinching a fold of skin, or by pricking the skin with a pin or point of a quill pen. The faradic current is a delicate method of comparing the sensitiveness of corresponding regions on the two sides. Faradic sensibility may be diminished when all other forms of cutaneous sensibility are normal. This is frequently observed in cases of sciatica; it occurs also in cases —such as lead paralysis—where there is well-marked motor paralysis.

The Rapidity of Sensory Conduction is determined by noting the interval between a prick and the signal given by the patient directly he feels it. In health the interval is much less than a second, but in locomotor ataxia there may be a long delay, even several seconds.

Sensibility to Temperature, often affected with the sensibility to pain, is most conveniently examined by applying hot and cold spoons to the part, or two test-tubes, one tube being filled with hot, the other with cold water.

Muscular Sense.—In addition to common sensibility, as illustrated by the pain of cramp, or that produced by squeezing the muscles, the

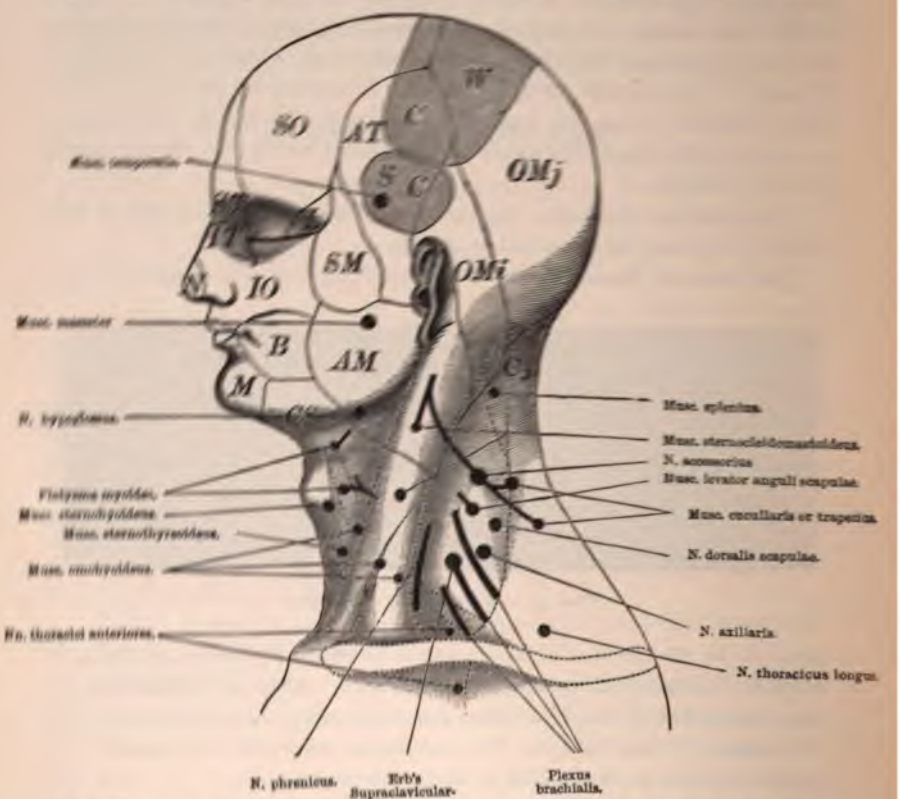


FIG. 233.—Distribution of the Sensory Nerves on the Head, as well as the Position of the Motor Points on the Neck. *SO*, area of distribution of supraorbital nerve; *ST*, supratrochlear; *IT*, infratrochlear; *L*, lacrimal; *N*, ethmoidal; *IO*, infraorbital; *B*, buccinator; *SM*, subcutaneous malæ; *AT*, auriculo-temporal; *AM*, great auricular; *OMj*, great occipital; *OMi*, lesser occipital; C_3, three cervical nerves; *CS*, cutaneous branches of the cervical nerves; *C*, *W*, region of the central convolutions; *SC*, region of the speech centre.

degree of contraction of the muscles is appreciated by the mind. This sense of muscular effort may be tested by placing various weights in a bag and suspending it to the part to be examined. In health, a difference of one-fortieth of the whole weight can be recognised.

Recognition of Posture may also be regarded as a part of muscular sense. It is tested by asking the patient, whose eyes are covered, to

move a limb into certain prescribed positions, thus to touch the tip of the nose with his index finger, to describe an imaginary circle with his great toe, or a limb is firmly grasped by the observer, and moved about in various directions, the patient being asked to indicate its position after it has been brought to rest. The muscular sense is often strikingly impaired in locomotor ataxia, also sometimes in hysteria and in cortical lesions.

Modifications of Cutaneous and Muscular Sensations, with their Diagnostic Value.—**Anæsthesia.**—The *degree* of anæsthesia,

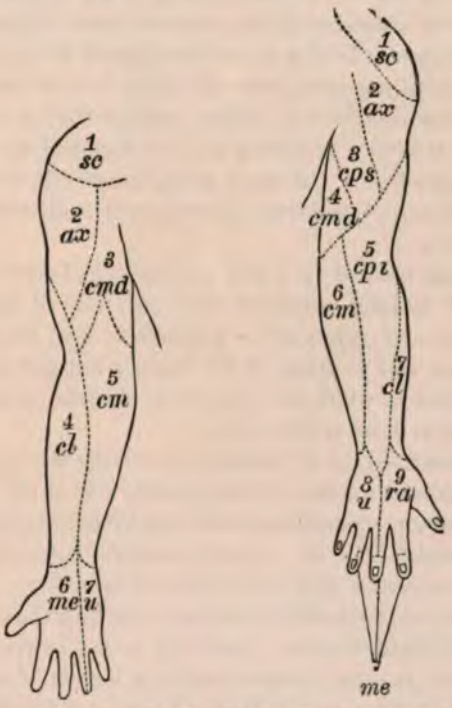


FIG. 234.—Distribution of the Cutaneous Nerves of the Upper Limb (*after Henle*). *Sc*, supra clavicular; *ax*, circumflex; *cmd*, area supplied by nerve of Wrisberg and intercosto-humeral *cps* and *cpi*, branches of musculo-spiral; *cm*, internal cutaneous; *cl*, musculo-cutaneous; *me*, median; *ra*, radial; *u*, ulnar.

whether partial or complete, its *variety*, whether limited to touch or pain, afford, as a rule, but little help in the localisation of a lesion. Complete anæsthesia of a part is more common in hysterical than in organic affections of the nervous system. In hysteria, too, the anæsthesia is often remarkable for the abruptness of its limitation and its want of correspondence to the anatomical distribution of the cutaneous nerves or spinal roots; thus it may affect one upper limb below the level

of a line drawn round the middle of the arm, or the whole of one side of the body, with the exception of the leg below the level of the patella. In syringo-myelia—a disease characterised by the association of muscular atrophy and anæsthesia in the upper part of the body—the loss of cutaneous sensibility is mainly confined to pain and temperature; occasionally, however, tactile sensibility is also involved. But in other diseases—as, for example, peripheral neuritis—the most frequent form of partial diminution of cutaneous sensibility is analgesia, with preservation of the tactile sense. "Anæsthesia dolorosa," in which an anæsthetic part is the seat of violent pains, if not of functional origin, is usually limited to affections of the posterior roots or peripheral nerves.

The most important feature to be investigated is that of *distribution*. When sensation is impaired over the lower half of the body and the lower limbs, the condition is called *paranæsthesia*; when over the lateral half of the body, including half the face and the extremities, it is called *hemianæsthesia*. In other cases, anæsthesia is limited to the area of distribution of particular nerves, or it is distributed in limited bands or patches.

Paranæsthesia testifies to a lesion of the spinal cord or of the cauda equina; and is usually associated with paralysis of the lower limbs. The distribution and variety of the paralysis is then the best indication as to the nature and position of the lesion; but the exact height on the limbs or body to which the anæsthesia extends furnishes a reliable guide to the upper level of the lesion.

Hemianæsthesia.—Loss of sensation involving the whole of one side of the body points to a lesion in the opposite side of the brain. When the loss of sensation is combined with impairment of the special senses, the lesion is situated in the opposite cerebral hemisphere, and most frequently at the hinder end of the internal capsule.

This condition of unilateral anæsthesia, affecting the special senses as well as the skin and mucous membrane as far as the middle line of the body, occurs also in extensive organic lesions of the cortex, but more commonly in hysteria (functional lesion of the cortex).

In hysterical hemianæsthesia, every form of cutaneous sensibility, as well as common sensation in the accessible mucous membranes, muscles, bones, and other deep structures, is more or less completely lost on the affected side, and even the muscular sense is often impaired or abolished. The special senses are likewise implicated; the senses of taste and smell are generally lost on the affected side, while hearing on that side is only impaired, and the affection of sight consists of a restriction of the fields of vision for form, and especially for colour, affecting to some extent both eyes, but most markedly the eye on the anæsthetic side.

Sometimes, in hysteria, anæsthesia of one side of the body is associated with hyperæsthesia and paralysis of the opposite side.

Hemianæsthesia of organic origin is usually less profound than that produced by hysteria, and when dependent on disease of the internal

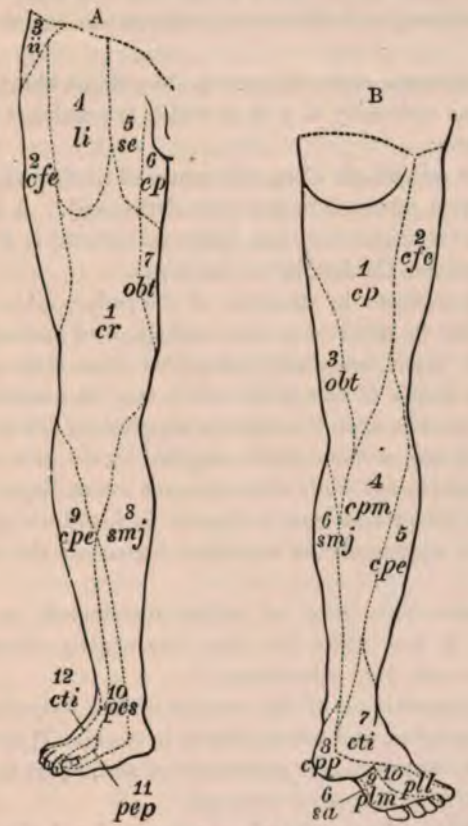


FIG. 235.—Distribution of the Cutaneous Nerves of the Lower Limb (*after Henle*). A. Anterior surface—1, anterior crural, middle and internal cutaneous branches; 2, external cutaneous; 3, ilio-hypogastric; 4, genito-crural; 5, ilio-inguinal; 6, posterior cutaneous and other branches of small sciatic; 7, obturator; 8, greater saphenous; 9, external posterior cutaneous; 10, musculo-cutaneous; 11, anterior tibial; 12, communicans tibialis. B. Posterior surface—1, posterior cutaneous; 2, external cutaneous; 3, obturator; 4, posterior cutaneous with filaments of tibial communicating; 5, posterior cutaneous branches of peroneal; 6, long saphenous; 7, communicans tibialis; 8, calcanean of posterior tibial; 9, internal plantar; 10, external plantar.

capsule, is found associated with hemianopsia instead of the visual defect just described as characteristic of hysteria.

Hemianæsthesia without disturbance of smell or sight may result from damage to the sensory tract between the pons and internal capsule. Hemianæsthesia with normal sensibility of the face, or a

crossed anæsthesia in which the face is affected on one side and the limbs on the other side, occur in lesions of the pons and medulla. Slight impairment of sensation on one side is not uncommon in hemiplegia of the ordinary type, but as a rule it is transient; whereas, when hemianæsthesia is distinct and permanent, there may be but little motor weakness, and this usually affects the leg more than the arm.

Limited Anæsthesia.—*Examples:*—1. The slight blunting of tactile sensibility at the extremity of a limb which is paralysed by disease of the cerebral cortex.

2. Patches of anæsthesia along the course of nerves whose roots are damaged by spinal caries or by tumours of the cord. A knowledge of the functions of the spinal roots (see tables and plates) is then essential in order to determine the locality of the lesion.

3. Areas of anæsthesia in affections of the peripheral nerves. Here it must be borne in mind that the conduction of sensory fibres in a mixed nerve is much less readily impaired than that of the motor fibres. Thus a lesion of the ulnar nerve may be unattended by any anæsthesia of the skin when the muscles supplied by it are more or less completely paralysed, or the muscles supplied by the fifth cranial nerve may be weak and wasted while the cutaneous sensibility of the face is unaffected, and yet post-mortem a tumour is found which compresses and flattens out apparently to an equal degree all the fibres of the nerve.

Hyperæsthesia.—This may be widely distributed, or limited to certain spots. It may affect the skin, the organs of special sense, the muscles, or other deep structures.

A *general* hyperæsthesia of the surface of the body is a prominent feature of hydrophobia, and occurs also in hysteria. Hyperæsthesia of one side of the body is a rare phenomenon, and, apart from hysteria, is chiefly met with in disease of the pons.

Of *local* varieties of hyperæsthesia, the narrow band of over-sensitive skin at the upper level of spinal lesions is one of the most common. It is most marked in cancer of the vertebræ, but is also usually well developed in caries of the spine, and in the various forms of spinal meningitis. The position of this zone is readily ascertained by pinching the skin along the sides of the trunk. It frequently encircles the body, extending forwards and a little downwards from one or more tender vertebral spines. When the spinal tenderness is not very obvious, it may be brought out by passing a hot sponge down the spine, or by the application of the kathode of a galvanic battery. When the posterior roots of the cervical or lumbar enlargement are implicated in a meningitis, bands of hyperæsthesia will be found on

those parts of the surface which are supplied by the irritated roots. Thus increased sensitiveness along the outer side of the upper limbs from shoulder to thumb points to irritation of the fifth cervical root. Hyperæsthesia of the skin and muscles is prominent in cases of neuritis; and an accessible nerve-trunk like the ulnar may be swollen as well as tender to pressure. In alcoholic multiple neuritis the muscles are often extremely sensitive, great pain, for example, being caused by slightly squeezing the calves or fleshy parts of the arms.

The pain of neuralgia is frequently accompanied by increased sensitiveness of the skin, more marked usually to tactile than to thermal impressions. The hyperæsthesia is commonly most intense at certain spots; these "*points douloureux*" usually correspond to a superficial part of the nerve, to its divisions, or to its union with another nerve-trunk.

Hyperæsthetic spots are also common in hysteria. Thus there may be tenderness of the spine or of the infra-mammary or hypochondriac regions, or over certain areas of the thorax or abdomen. Of the latter, the most frequent and characteristic are the ovarian regions, where tenderness may be superficial or deep. Pressure over the ovarian regions, or indeed sometimes over hyperæsthetic spots in any part of the body, may cause great distress and give rise to fainting, to globus, or even to convulsive attacks; hence these hyperæsthetic areas have been called "hysterogenic." The local tenderness of the dorsal spine in hysteria may be contrasted with that around the sides of the body in vertebral caries.

Paræsthesia.—This term includes:—

1. The presence of abnormal sensations in the absence of any outward stimulus. Thus (*a.*) subjective tactile sensations, as formication, a feeling of creeping, as if ants were crawling over the skin; itching and the like. (*b.*) Subjective painful sensations, such as stinging, pricking, or smarting. (*c.*) Subjective sensations of heat and cold.

2. Perversion of the cutaneous sensibility—that is, the production by an external stimulus of a feeling different from that experienced in health. Thus a touch of the finger may give rise to pain, a prick to a burning sensation, the application of cold to a stinging sensation. Sometimes a single touch is felt as two, three, or even five points; this is called **polyæsthesia.** In other cases a patient is unable to tell what part is touched, and he may refer an impression on one side to a corresponding place on the opposite side of the body—then the condition is named **allocheiria.**

The above modifications of normal sensation may occur from disease

[illegible]

[illegible]

[illegible]

[illegible]

Reflex Disorders of Muscles.—Irritation of a sentient surface may cause an alteration in the strength of a normal reflex movement, or it may lead to changes, temporary or permanent, in the tone or nutrition of muscles. **Reflex movements** may be produced in health either by stimulation of the skin or accessible mucous membranes, or by excitation of tendons, fascia, or periosteum. The former are called *superficial*, the latter *deep* reflexes.

The **Superficial Reflexes** include the cutaneous and the cranial

reflexes. The **cutaneous reflexes** consist of quick muscular contractions, caused by tickling the skin with a feather or the finger, or by scratching or tapping it sharply. The following (tabulated after Gowers) may be readily obtained in health, especially in young children, and alterations of their natural vigour are often of great significance in disease :—

Name of Reflex.	Mode of Excitation.	Nature of Result.	Level of Cord upon which Reflex Depends.
Plantar.	Stroking sole of foot.	Movements of toes; of these and foot; or of these and leg.	Second sacral nerve (lower part of lumbar enlargement).
Gluteal.	Stroking skin of buttock.	Contraction of glutei.	Fourth and fifth lumbar nerves.
Cremasteric.	Stimulation of skin at upper and inner part of thigh.	Retraction of testicle.	First and second lumbar nerves.
Abdominal.	Stroking abdominal walls downwards from costal margin to nipple-line.	Contraction of upper or of lower part of abdominal muscles.	Eighth to twelfth dorsal nerves.
Epigastric.	Stroking side of chest downwards from nipple.	A dimpling of corresponding side of epigastrium.	Fourth to sixth or seventh dorsal nerves.
Scapular.	Irritation of interscapular region.	Contraction of scapular muscles and of portion of axillary fold.	Sixth cervical to second dorsal nerve.

Clinical Value.—In forming an opinion of the vigour or feebleness of the cutaneous reflexes, it should be borne in mind (1) that they vary much in different normal individuals; (2) that they are usually more marked in children than in adults, and in women than in men; (3) that it is often difficult to elicit the abdominal reflex when the belly is large and flaccid, and that the cremasteric reflex is often absent in elderly men; and (4) that occasionally repeated trials fail to produce some of the reflexes, as the gluteal and scapular, even when there is no reason to suspect disease of the nervous system.

Keeping in view these precautions, the following conclusions may be drawn :—

The presence in average strength of a cutaneous reflex indicates that the reflex arc upon which it depends is intact and probably healthy.

Exaggeration of a cutaneous reflex suggests either that some part of the arc is irritated or that it is separated from the control of a higher centre. Thus the reflexes are increased when the grey matter is unduly stimulated, as in tetanus and strychnine poisoning, or in conse-

quence of irritation of the posterior and anterior roots (the afferent and efferent portions of the reflex arc respectively), as in cases of pachymeningitis. Commonly, too, they are exaggerated when there is disease of the cord above their level; sometimes there is an initial inhibition of reflexes, which subsequently are found to be increased. A lasting diminution or absence of cutaneous reflexes below the level of the original lesion is usually attributed to downward extension of the disease in the cord, but Bastian holds that a complete transverse myelitis, say of the cervical region, will of itself sometimes cause abolition instead of exaggeration of the reflexes at a lower level.

Diminution or absence of a cutaneous reflex points to a defect at some part of the reflex arc.[1] Thus, in infantile paralysis affecting one of the lower limbs, the cremasteric reflex may be lost while the plantar is retained, because the upper but not the lower part of the lumbar enlargement is diseased. In dorsal myelitis the condition of the reflexes often affords valuable information as to the seat of disease; thus, if the epigastric reflex is normal and the abdominal absent, we may assume that the cord is affected between the sixth and tenth dorsal nerves. As a rule, in myelitis, and also in locomotor ataxia, the degree of enfeeblement of the reflexes varies with that of tactile sensibility, although sometimes it will be found that the reflexes are lost above the line of anæsthesia. In disseminated sclerosis loss of the abdominal reflexes is sometimes a valuable indication that the grey matter of the cord has become involved. In hemiplegia of organic origin the superficial reflexes are usually enfeebled or lost on the paralysed side, but in hysterical hemiplegia they are generally normal, with the exception of the plantar reflex, which is frequently lost. Loss of the plantar reflex is also a characteristic feature of damage to the cauda equina.

The chief **cranial reflexes** are:—

1. The closure of the eyelids caused by irritation of the conjunctivæ.
2. The contraction of the pupil to light, or its dilation by irritation of the skin of the neck.
3. Spasm of the facial muscles by irritation of the fifth nerve.
4. Sneezing and lacrimation by irritation of the nasal mucous membrane.
5. The contraction of the palate by irritation of the fauces.

The palate reflex is enfeebled or lost in hysteria, also in bulbar paralysis.

The pupil reflexes are considered under the examination of the eye.

[1] Diminution of a reflex is shown not only by its weakness and by the difficulty in obtaining it, but also by the slowness of the movement.

The Deep Reflexes.—The Knee-Jerk or **Patellar Tendon Reflex** is the forward jerk of the foot and leg, which is produced by tapping the ligamentum patellæ with the tips of the fingers, the inner border of the hand, a percussion hammer, or the edge of the ear-piece of the stethoscope. If a doubtful result is obtained, the knee should be laid bare. The jerk is caused by sudden contraction of the quadriceps, and it is essential that this muscle should be stretched to a certain extent. This is usually obtained by crossing the leg to be tested over the other; the knee of the supporting leg being at a right angle. Another

FIG. 236.—Method of obtaining the Knee-jerk.

convenient posture is to get the patient to sit on the edge of a table, or, if a child, on the edge of a chair.

In stout persons who cannot cross one knee over the other except in a stiff fashion, the operator may support the limb to be examined by passing his hand beneath the patient's thigh and grasping the opposite knee. Some patients appear to have great difficulty in keeping the knee loose; in such cases the muscular tension may often be overcome by getting the patient to hold up his head, open his mouth, or put out his tongue; and it is also helpful for him to interlock the bent fingers

of each hand, and to pull strongly while the ligamentum patellæ is being struck (see Fig. 236). When the patient is confined to bed, the knee should be slightly flexed and the thigh supported just above it. It is also frequently useful to push down the patella with one hand, while the patellar tendon is struck with the other.

In health, the facility with which the knee-jerk may be obtained varies in different persons, but to a less extent than the cutaneous reflexes. It is commonly more sluggish in the child than in the adult, but is more active in infancy, and is readily obtained at birth. It is often difficult to elicit in elderly persons.

Exaggeration of the knee-jerk occurs: (1.) In phthisis and other exhausting or febrile diseases. (2.) In hysteria and in chronic rheumatism. (3.) In tetanus and in poisoning by strychnine. (4.) It is a marked feature of all forms of spastic paralysis; thus, in hemiplegia the knee-jerk is increased on the paralysed side, and often, too, on the other side; in lateral sclerosis, whether primary or as a part of disseminated sclerosis, or of amyotrophic lateral sclerosis. In the last-mentioned disease exaggeration of the reflex is found in association with muscular atrophy. Occasionally muscular spasm is so extreme that the knee phenomenon cannot be properly elicited; this is sometimes the case in the spastic hemiplegias of infancy. (5.) It is not uncommon in the early stage of peripheral neuritis.

Enfeeblement or absence of the knee-jerk occurs whenever any portion of the reflex arc is interrupted, thus:—

(1.) In locomotor ataxia, of which disease it is an early and important sign. (2.) In atrophic paralyses, whether of spinal or neural origin, as infantile paralysis, alcoholic neuritis; often, too, in diabetes and diphtheria. In descending myelitis it sometimes constitutes a valuable premonitory symptom of approaching bladder disturbance, for if it be noticed that the muscles on the front of the thigh are getting weak and wasted, and that the knee-jerk is lost, it is highly probable that paralytic incontinence of urine will soon appear. (3.) In pseudo-hypertrophic paralysis. (4.) In some cases of cerebellar tumour, probably when one of the lateral lobes is affected. (5.) During and for a short time after the convulsive stage of an epileptic attack; also in coma from any cause.

When the knee-jerk is absent, a slight hollow over the patellar tendon may often be observed; the percussing fingers do not feel that sense of resistance which is always experienced when the knee-jerk is present, and which is a valuable sign in cases where, from obesity or other causes, there is little or no movement of the leg; we feel convinced from the sense of resistance that the reflex exists, but are unable to demonstrate its presence, and that our opinion is correct is

often proved by the fact that a satisfactory knee-jerk is obtained on the next examination.

Wrist and Elbow Jerks.—A tap with the edge of a stethoscope over the lower end of the radius produces flexion of the elbow in many healthy persons. The movement is usually due to contraction of the supinator longus, but if the reflex is exaggerated the other flexors of the elbow are brought into action. Flexion of the fingers may also occur, but as a rule this movement is better produced by tapping over the front of the wrist.

Contraction of the triceps is caused by tapping over its tendon just above the olecranon, and sometimes by tapping over the lower end of the ulna.

These reflexes at the wrist and elbow are due to stimulation of afferent nerve fibres in connection usually with tendons, but sometimes with fasciæ or periosteum. In the latter case they are called **periosteal** or **fascial reactions.** Sometimes it is difficult to say whether a movement is due to tapping over periosteum or over tendon. For example, a tap over the lower end of the radius affects in some cases the periosteum, in others the tendons passing over it. In the former case flexion of the elbow is commoner than flexion of the wrist or fingers, but in the latter the reverse holds.

The reflexes referred to have a like pathology to that of the knee-jerk; thus, they are increased in cases of spastic paralysis, *e.g.*, in hemiplegia and in lateral sclerosis; they are diminished or abolished in cases of atrophic paralysis, *e.g.*, in peripheral neuritis. Curiously enough, however, the wrist-jerk is exaggerated in many cases of wrist-drop from lead poisoning. But it must be noted that the intensity of the reflex movements obtained at the wrist and elbow vary in health much more than the intensity of the knee-jerk; thus in many persons the wrist-jerk is completely absent, while in others it is present in great excess.

Occasionally their diagnostic value is great. For example, a patient suffers from paresis, in association with some slight sensory disturbance, of the distal portion of one upper limb. The condition may be due to peripheral neuritis, or to a lesion in the cortical centre for the arm. Now in such a case, exaggeration of the wrist and elbow jerks as compared with the other arm would be strong evidence in favour of a cortical lesion, while their absence would point to peripheral neuritis.

Precaution.—Irritability of muscular tissue, as shown by a marked contraction produced by tapping over the belly of a muscle, must be carefully distinguished from exaggeration of the deep reflexes. The pathological significance of the former condition is not clearly made

out. Sometimes it is present when the deep reflexes are in excess, as in advanced phthisis; in other cases muscular irritability is associated with loss of the deep reflexes, thus in locomotor ataxia, tapping over the quadriceps femoris may cause its vigorous contraction in cases where the knee-jerk is entirely absent. In all probability the condition referred to is more commonly met with in cases of atrophic than of spastic paralysis.

Ankle Clonus or **Foot Clonus** is usually found in association with marked exaggeration of the knee-jerk; it is but rarely present in health, and then only in a modified form, thus tapping on the tendo Achillis may cause contraction of the calf muscles, together with sudden extension of the ankle.

To elicit ankle clonus, the knee should be slightly flexed; the fore part of the foot is then lightly grasped and suddenly and forcibly pressed upwards towards the tibia. Immediately the calf muscles contract and depress the foot; the muscles now relax, and the pressure of the hand being continued, the foot is once more dorsally flexed, when the calf muscles again contract and extend the foot a second time, and the clonic series of spasmodic contractions continues so long as the tension of the tendo Achillis is maintained. The movements are uniform, and occur from six to nine times every second.

Ankle clonus may be produced by any of the conditions which lead to increase of the knee-jerk. Occasionally, as in some cases of disseminated sclerosis, it may be developed when the patellar reflex is not in excess, or even when this is absent, but a converse condition is more frequent; and in children, even when the knee-jerk is greatly increased, it is rare to obtain ankle clonus.

Both ankle clonus and exaggerated tendon reactions may be found in typhoid and other febrile disorders.

A loose kind of ankle clonus, consisting of a few imperfect movements, and differing from the typical clonus met with in spastic paralysis, may often be developed in cases of hysteria and neurasthenia, but it is commoner to find irritable knee-jerks and no ankle clonus. Occasionally well-marked ankle clonus is obtained in hysteria.

Toe Clonus.—When there is increased tension of the foot muscles, sudden passive extension of the first phalanx of the great toe may produce rhythmical flexion of the toe by the contractions of the abductor and flexor brevis.

Wrist Clonus.—Pressing the hand backwards into a hyper-extended position will, in certain conditions, as the late rigidity of hemiplegia, excite movements like those of ankle clonus.

Spinal Epilepsy is a term applied by Brown-Séquard to paroxysms

of violent tremors which sometimes affect the lower limbs in cases of spasmodic paralysis. The clonic spasms may often be arrested by grasping the toes of one foot and bringing them suddenly and forcibly into plantar flexion.

Paradoxical Contraction is a name which was given by Westphal to a slow tonic contraction occurring in a muscle when suddenly relaxed or shortened. This phenomenon is best seen in the tibialis anticus, which, under certain ill-ascertained circumstances, contracts when the foot is grasped and dorsally flexed; the tendon of the muscle stands out, and the foot may remain stiffly flexed for some minutes. This anomalous reaction has been observed in the early stage of locomotor ataxia and in some other nervous affections, but appears to have no affinity with exaltation of the deep reflexes.

Alterations in the Strength, Tone, or Nutrition of Muscles of Reflex Origin.—Transient paralysis sometimes appears to be due to peripheral irritation. *Examples*:—Inability to pass urine after an operation on the anus, such as the removal of piles or the division of a fistula; transient weakness of one arm after opening the chest for empyema; transient ptosis, which very rarely follows irritation of the fifth nerve, as from extraction of a tooth.

Muscular Spasm.—The spasmodic condition of the muscles in cases of spastic paralysis probably largely depends on external stimulation of the sensory nerves; and the author believes that, very rarely, intense spasm of the legs may be started by peripheral irritation, and may become a permanent condition apart from any evidence of paralysis or other indication of disease of the nervous system. Of examples of spasm affecting the involuntary muscles may be mentioned intestinal colic from irritation of the mucous membrane of the bowel, and attacks of asthma following irritation of the nasal mucous membrane. General convulsions, too, are frequently induced in young children by peripheral irritation, as by the presence of a worm in the intestines.

Muscular Wasting.—This, as already pointed out, frequently supervenes on injury or disease of a joint, and there is some evidence in favour of its being a reflex phenomenon.

LANGUAGE AND ITS DISORDERS.

Communication between human beings by means of language is effected by an outgoing and an ingoing mechanism, with their connecting links.

The Outgoing Mechanism is constituted by cortical centres, the cells of which are connected by nerve-fibres with similar groups of cells or nuclei in the medulla or spinal cord, whence nerve-fibres proceed to

supply the muscles used in articulation, vocalisation, writing, and various gestures.

The Ingoing Mechanism, constituted by the eye and ear, and in the blind by the sense of touch, together with their sensory paths and centres, is made use of when we try to comprehend the language of another by listening to his utterances, by reading his writing, or by watching his gestures.

Disorders of language, therefore, may be classed into motor and sensory varieties. Thus, just as a limb may be paralysed either as regards motion or sensation, so speech may be affected by a motor or a sensory paralysis.

Vocal Speech is composed of two elements, phonation and articulation. *Phonation* is produced in the larynx, and its defects are referred to in the section on the larynx. *Articulation* is produced in the cavity of the mouth by the muscles of the lips, tongue and palate, and these, just like the muscles of a limb, may be affected by spasm, tremor, incoördination, or paralysis: and it is of great importance to recognise that the movements concerned in articulate speech are dependent, just as much as those of a limb, on a motor mechanism composed of cerebral centre, conducting path, nuclei of origin in the medulla and cord, motor nerves and muscles, and that the spasm, tremor, incoördination or paralysis which may affect alike speech or a limb are produced by damage to some part or parts of the motor mechanism. A thorough comprehension of this fact will alone give the student an adequate conception of impaired speech processes, especially of those that result from damage to the higher or cerebral mechanism.

Disorders of the Outgoing Mechanism—Articulate Speech.—**Stammering** or stuttering is a spasmodic disorder of speech, which chiefly and most commonly affects the muscles of articulation, but may involve those of the glottis, or even those of respiration. The check in enunciation usually manifests itself in the pronunciation of the explosive consonants, *b*, *p*, *d*, *t*, hard *g*, and *k*, but may occur during the production of other consonants, and even of vowel sounds, and, in exceptional cases, the sufferer stammers while whispering or singing. The check is followed by a painful pause, during or after which the sound at which the hitch occurs is rapidly repeated, until the complete word is uttered. Sometimes the patient is quite unintelligible. There is a tendency for other groups of muscles to become involved, so that in severe cases the mouth may remain open, while the muscles of expression are convulsed, the glottis is contracted, respiration is arrested and violent spasms affect the limbs and trunk.

The defect usually appears between the age of four years and puberty, but it may come on at any age. Sometimes it follows febrile or func-

tional nervous disorders; sometimes it is the result merely of temporary debility.

Aphthongia is a term applied to temporary spasms affecting the muscles supplied by the hypoglossal nerves, the spasms being brought on by any attempt to speak.

The difficulty of speech in chorea also depends on spasmodic movements of the tongue and mouth, and sometimes also on spasm of the respiratory and laryngeal muscles. Thus a sudden, deep, or jerky inspiration will often cut off the last syllable of a word; words are uttered too quickly, or with a drawl or hesitation.

Tremor of the muscles of articulation is seen in general paralysis of the insane, in meningitis, in disseminated sclerosis, and in delirium tremens.

Scanning, Staccato, Syllabic Speech.—This is characteristic of disseminated sclerosis. In a typical case a pause occurs after each syllable, the syllables are slowly evolved and are unduly accented. The patient appears to speak with effort, and tremor of the lips and tongue is often present. A somewhat similar defect may accompany locomotor ataxia.

Slurred or Clipped Speech occurs in general paralysis of the insane. Words are slurred over and uncompleted, or "clipped;" there is tremor, and often a peculiar pouting of the upper lip, which seems to be stiff and as if glued to the lower lip, while the effort to articulate brings into prominence the want of control over the lower facial muscles.

Other Defects in Articulation, comprised under the terms **anarthria** or **dysarthria**, are most prominent in affections of the medulla and lower part of the pons. The tongue usually suffers first, and hence *l*, *r*, *n*, *t*, are imperfectly pronounced; when the lips become paralysed, the letters *o*, *p*, *b*, *m*, are indistinct; paralysis of the palate gives a nasal quality to the voice, *p* and *b*, for example, being sounded like *m* and *v*. Finally, in some cases of bulbar paralysis, there is total inability to articulate a single word, a condition called **alalia**; then, too, the power of swallowing is lost, and saliva cannot be retained in the mouth. **Paralalia** is sometimes used to express an inability to pronounce words correctly; thus, in ordinary hemiplegia or in paralysis of the facial nerve, the utterance is often thick, and difficulties of articulation may also be present in abnormalities affecting the larynx, nose, palate, teeth, or lips.

The defects of speech alluded to above depend for the most part on imperfect action of the muscles of articulation, due either to disease of the muscles, their nerves, or nuclei of origin. But under the name **Aphasia** we refer to disorders of speech which result from lesions of

the cerebral hemispheres, and which are due either—(1) to paralysis of the special movements of articulation, or of the special movements of the hand used in writing; or (2) to paralysis of the receptive sensory centres. The former class constitutes motor aphasia, the latter sensory aphasia. There are also mixed cases, which form a class of combined motor and sensory aphasias.

Motor Aphasia.—In complete motor aphasia there is inability to

FIG. 237.—The Co-ordinated Cortical Mechanism for Speech Processes. (*After Young and Ross.*) From the eye and ear centripetal fibres (*v* and *a*) ascend to terminate in the angular gyrus (V) and first temporo-sphenoidal convolutions (A) respectively, but in reality these fibres are directly connected with a much larger area of the cortex than is here indicated. In addition to these, fibres of muscular sense (*s*, *s'*, and *s''*), indicated by dotted lines, ascend from the muscles of articulation, from those of the hand and from those of the eyeball to reach the cortex. The centres of vocal and written expression are represented at E and W, and these are connected by means of centrifugal fibres, *m* and *m'*, with the vocal apparatus and hand respectively. ("*Aphasia*," by Ross.)

speak, called **aphemia**, to write, **agraphia**, and there may be loss of power to express thoughts by signs or pantomime, **amimia**. The essential peculiarity is the *loss of voluntary speech*, the patient, while understanding everything that is said to him, and while retaining more or less his previous powers of understanding written words, is completely incapable of repeating a single word spoken to him. But, although *speechless*, he is not always *wordless*—that is, he often articulates

certain words, which are usually the same for the same patient, and have therefore been called "*recurring utterances;*" they are commonly either the words actually spoken, or those about to be spoken, when the damage to the brain occurred. Thus the recurring utterance of a woman who was taken ill in a cab after telling the cabman to drive her to Mrs. Waters was "Misses" (Gowers); that of a librarian was "List complete" (Russell); that of a girl attacked when riding on a donkey, "Gee, gee" (H. Jackson).

Words or phrases of this kind must be regarded as of an interjectional or emotional character rather than as expressions of an intellectual state.

It is also to be observed that the patient may be aware of his errors in speaking, that what utterance he has is usually clear and distinct, and that in singing he may be able to utter every word of a song although unable to speak it. Every gradation may be met with between the most severe degree of aphemia, in which the patient replies to questions by grunting noises or meaningless syllables,—being practically wordless as well as speechless,—and very slight defects, where the paralysis of speech is only indicated by a hesitating slow utterance or by slight mistakes in the use of words. The chief varieties of aphemia are represented in the following table, taken from Dr. Ross's book on "Aphasia":—

APHEMIA.

The Faculties of Spoken Speech.	Varieties.				
	1st deg.	2nd deg.	3rd deg.	4th deg.	5th deg.
1. Spontaneous vocal speech in sentences.	Impaired.	Lost.	Lost.	Lost.	Lost.
2. Repetition of words and reading aloud.	Retained.	Retained.	Lost.	Lost.	Lost.
3. A few intelligent replies to questions in single words.	Retained.	Retained.	Retained.	Lost.	Lost.
4. Occasional and recurring utterances of no speech value.	Retained.	Retained.	Retained.	Retained.	Lost.
5. Grunting sounds and syllabic utterances, not forming any word.	Retained.	Retained.	Retained.	Retained.	Impaired.

Permanent motor aphasia results most frequently from a lesion in the *posterior part of the third left frontal convolution*, but it may depend on a lesion in the centrum ovale underlying this convolution, although, if the motor path is involved some distance below the cortex, the defect of speech is transient and soon recovered from. It must be remembered, too, that most cases of ordinary, right-sided hemiplegia, due to damage to the posterior half of the internal capsule, are accompanied by a temporary motor aphasia.

It is probable that the centre for writing occupies the posterior part of the *second* left frontal convolution.

There are also many degrees of **motor agraphia**. The patient, if his hand is not paralysed, may be able to copy words or sign his name when he has lost the power of expressing his thoughts in writing, but even when the disability is slight the spontaneous writing of simple sentences presents egregious blunders in spelling and diction. Rarely agraphia may exist without aphemia.

MOTOR AGRAPHIA.

The Faculties of Written Speech.	Varieties.				
	1st deg.	2nd deg.	3rd deg.	4th deg.	5th deg.
1. Spontaneous writing in sentences.	Impaired.	Lost.	Lost.	Lost.	Lost.
2. Writing to dictation and copying sentences.	Retained.	Retained.	Lost.	Lost.	Lost.
3. Writing and copying imperfectly a few single words.	Retained.	Retained.	Retained.	Lost.	Lost.
4. Writing letters of the alphabet.	Retained.	Retained.	Retained.	Retained.	Lost.
5. Copying geometrical figures.	Retained.	Retained.	Retained.	Retained.	Impaired.

Disorders of the Ingoing Mechanism.

1. **Damage to the Ingoing Current.**—Deaf-mutism, in which a person cannot speak because he cannot hear, is an example of damage to the ingoing current along the auditory nerve. Thus a child born deaf never learns to talk, and if a child under seven years loses its hearing in consequence of disease—as from a lesion of the labyrinth—its speech gradually deteriorates, and it may become completely dumb.

2. **Damage to Sensory Centres—Sensory Aphasia.**—The

simplest varieties, though rare, are afforded by uncomplicated cases of word-blindness and of word-deafness.

Word-Blindness.—A patient suffering from this disorder is unable to read printed or written words, and in some cases he cannot recognise a single letter. He sees the letters and words, and may read aloud, after making elaborate preparations, but there is no correspondence between the actual and the spoken words. He may, however, be able to recognise portraits and simple geometrical figures, to tell the time by a watch correctly, and often he correctly names all objects presented to him. His power of writing, if not entirely lost, is usually limited to short words, written either spontaneously or to dictation, and he often writes better when his eyes are closed than when they are open.

In an example of pure word-blindness there is no defect in spoken speech, the patient can repeat correctly all words uttered in his hearing, he also understands all that is said to him, and answers questions correctly.

In association with word-blindness there is almost invariably some degree of right bilateral hemianopsia. The lesion most commonly implicates the lower and hinder part of the left parietal lobe (including the angular gyrus), together with the adjacent portion of the occipital lobe.

Word-Deafness.—In uncomplicated cases of word-deafness the patient can speak, and, if an educated person, may be able to read intelligibly; he hears sounds as well as before his seizure, but he fails to understand spoken language. An uttered request to close his eyes or to hold out his hand, when unaccompanied by expressive gesture, is not attended to; he hears that some one is speaking, but the words do not revive corresponding ideas. Objects presented to him are also named incorrectly (this is called the **aphasia of recollection**). There is also inability to write from dictation, and in some cases to repeat words. Strangely enough, sometimes a complicated sentence is better understood than a simple one.

Examples of pure word-deafness are rare, for language being first developed in connection with the auditory mechanism, any damage to the auditory speech-centre (the first and second temporo-sphenoidal convolutions) is likely to interfere with the other departments of speech. Thus, not only is some degree of word blindness usually present, but motor speech processes are also affected; to these disorders, in cases of sensory aphasia, the term **paraphasia** was applied by Kussmaul. A slight degree of paraphasia may be temporarily evinced by a nervous or an exhausted person, as when on meeting a friend he calls him by a wrong name. A worse degree is observed in cases

of word deafness, when all objects are named wrongly, as *worm powder* for *cough medicine*, *parasol* for *castor oil*. In still worse cases all objects are called by the same name; thus a patient under the care of Dr. Ross, who previous to his attack had been a heavy drinker, called his finger, or any other object presented to him, a public-house. In the worst variety of paraphasia, speech is mere inarticulate jargon, and the condition is called **gibberish aphasia**. Such an aggravated condition, occurring either in connection with word blindness or word deafness, is nearly always associated with hemiplegia, and cases presenting such marked motor defects are to be regarded as examples of a mixed motor and sensory aphasia. In sensory aphasia mistakes in writing corresponding to those made in speaking are also frequent, and are classed under the term "**paragraphia**." The patient writes wrong words, makes errors in spelling simple words, is perhaps unable to complete a sentence in writing, and if severely affected he cannot form a single intelligible word.

Disorders of the Connecting Links.—The cortical centres for speech are assumed to be healthy, but the fibres connecting them are diseased.—**Verbal Amnesia, or the Aphasia of Recollection**, the third simple variety of sensory aphasia, is always present to some extent in cases of word blindness or word deafness, but may exist alone.

In this condition there is a loss of memory for words. Most persons have experienced a slight degree of this in temporarily forgetting the name of a friend, or of some familiar object; in worse cases there is an inability to name most of the objects by which the patient is surrounded, although, if the name of an object is written down or uttered in his hearing, he immediately associates the name with the particular object in question.

It will be gathered from the above necessarily brief account of aphasia:—

(1.) That cases of sensory aphasia usually present a combination of the three simple varieties in varying proportions—pure examples of word blindness, or of word deafness or verbal amnesia being rare.

(2.) That some motor disorder of speech is of necessity associated with every sensory disorder.

It is therefore essential, in the investigation of any form of aphasia, to first ascertain whether it is the motor or the sensory nervous mechanism of speech that is *chiefly* affected; and if the aphasia is mainly sensory, to determine to which variety it mainly belongs, ignoring for the time any peculiarity in motor disorder. To this end the following tests may be employed:—

1. Ask the patient to perform some simple action, such as "Hold

out your hand," "Close your eyes," being careful not to convey information by facial expression or gesture. If he does not carry out the request, and if he does not appear to understand any simple question, he has word deafness. It is also useful to ask him some ridiculous question, such as "Are you a hundred years old?" If the question is not emphatically denied by speech or gesture (the patient giving evidence that he hears ordinary sounds), we may be quite sure that he is word deaf, and that there is disease of the first temporal convolution, or immediately beneath it.

2. If word deafness is excluded, try him with similar requests in writing; ascertain, also, whether he can read short words. Before, however, deciding that the patient is unable to understand written language it is desirable to write down the name of some simple object, such as *pen* or *key*, and then bring before him several objects, including those written, and see whether he is able to associate the written name with the actual object. If he cannot do this, and if it be also found that there is hemianopsia, the patient is suffering from word blindness.

3. The presence of motor aphasia is established by finding that the patient, while giving prompt obedience to written or spoken request, is unable to give pertinent replies to questions, and generally also to repeat words uttered in his hearing, or presented to him in writing.

DISORDERS OF VISION, INCLUDING CHANGES IN THE EXTERNAL APPEARANCE AND MOVEMENTS OF THE EYE.

In addition to a description of disorders of vision, which depend upon disease of the nervous system, it is convenient to include a brief summary of other morbid conditions of the eye, in so far at least as they form part of or are caused by general diseases. Both classes may be grouped together and considered in the following order:—

1. Affections of the Conjunctiva.
2. Affections of the Cornea and Iris.
3. Disorders of Muscular Action.
   - (1.) Of Internal Muscles.
   - (2.) Of External Muscles.
4. Amblyopia and Functional Disorders of Sight.
5. Changes detected by means of the Ophthalmoscope.

Affections of the Conjunctiva.—The chief changes to be noticed are yellowness, œdema, signs of congestion or inflammation, subconjunctival hæmorrhage.

Yellowness.—Yellowness of the conjunctiva is one of the earliest and most constant signs of jaundice. Care must be taken to distinguish it from the yellowish appearance due to other causes; thus the conjunctiva often appears yellow in pernicious anæmia, owing to the shining through of the subconjunctival fat. A yellowish discoloration, with enlargement of the conjunctival vessels, is common in chronic alcoholism and in other cases when venous congestion is present. The **brownish** discoloration caused by long-continued application of nitrate of silver may here be mentioned.

Œdema.—Slight œdema of the conjunctiva causes the watery and glistening eye which is frequently observed in Bright's disease. When œdema is very great, it constitutes *chemosis*. This is noticed in purulent ophthalmia and accompanies severe inflammation of surrounding parts.

Congestion.—Apart from local conditions, congestion of the conjunctiva may occur in several general diseases, of which may be specially mentioned: common catarrh, measles, typhus, heart disease, rheumatic fever. Frequently, too, it is the result of severe coughing.

Inflammation.—Acute forms of conjunctivitis, of varying intensity, and characterised by redness, swelling, and more or less muco-purulent discharge, are met with in small-pox, measles, hay-fever and gonorrhœa. The condition occurs also as a result of direct irritation. *Membranous* ("*Diphtheritic*") *Ophthalmia.*—The ophthalmia of measles, and sometimes of other infantile diseases, occasionally takes on a membranous type, the membrane being limited almost invariably to the palpebral conjunctiva. A very severe form, in which the whole thickness of the conjunctiva is involved, and the lids are much swollen, has been observed as an effect of the true diphtheritic poison in a few cases.

Subconjunctival Ecchymosis.—This is of a purplish hue, and occurs as a result of straining efforts, as in severe coughing or vomiting. Ecchymotic patches in the conjunctiva are also sometimes met with in severe forms of acute ophthalmia.

Affections of the Cornea.—For clinical purposes, these may be divided into *Opacities* and *Ulcers*. They not infrequently coexist; in fact, a certain amount of opacity, due to infiltration with inflammatory products, is nearly invariably found round an ulcer. Many of these affections, especially when acute, are accompanied by "*ciliary congestion.*" This is indicated by a ring surrounding the cornea, composed of closely set, straight, radiating vessels. It is of a pink colour, duller than the bright red of conjunctival congestion and inflammation. This is also a prominent symptom of iritis.

The chief *opacities* of the cornea are:—

Arcus Senilis and Inflammatory Arcus.
Nebula and Leucoma.
Steamy Cornea.
Opacity from Lead.
Calcareous Film.
Pannus.
Ground-Glass Cornea.
Dotted Opacity.
Onyx and Hypopyon.

The last three of these are associated with, more or less, "ciliary congestion."

Arcus Senilis.—This is a white or yellowish opacity at the junction of the cornea and sclerotic. It commences under the upper lid in the form of a crescent; then it affects the lower margin of the cornea and, at a later period, may encircle the whole cornea. True arcus is rarely met with before the age of forty years, but opacities bearing a superficial resemblance to it, but whiter and of more irregular outline, may occur at an earlier age, as a result of inflammation in the neighbourhood.

Nebula and Leucoma.—These are names given to opacities of the cornea the result of injuries or of inflammatory processes. They are often valuable indications of previous disease, especially of interstitial keratitis and strumous ulceration. When faint, and forming a mere cloud, the opacity is called a nebula; when dense and opaque white, it is called a leucoma. A leucoma to which the iris has become adherent is called *Leucoma adherens.* It indicates that there has been an ulcer which has perforated into the anterior chamber. It is to be remembered that a nebula so faint as to be very difficult to detect may cause a very serious defect of vision.

Steamy Cornea.—A slight degree of superficial opacity, so that the cornea presents the appearance of glass which has been breathed upon, is common in the earlier stages of corneal inflammations. A uniform steaminess is one of the prominent symptoms of glaucoma.

A characteristic opacity sometimes follows the application of lead lotion to a cornea the surface of which is abraded. This is a sharply defined, opaque, pure white spot, due to the deposition of lead salts in the corneal tissue.

Calcareous Film of the cornea is a transverse band of a greyish colour, containing white calcareous deposits. It occurs chiefly in eyes that have long been lost from some severe inflammatory process.

Pannus is an opacity with vessels extending into it from the conjunctiva. It occupies the part of the cornea covered by the upper lid, or sometimes all the cornea. It is a concomitant of trachoma.

Ground Glass Opacity (Interstitial Keratitis).—Here the whole thickness of the cornea becomes hazy and the surface steamy, so that the cornea bears some resemblance to ground glass. The opacity differs in intensity at different parts, so that it looks patchy on close inspection. It may be so dense that the iris can with difficulty be discerned. Straight vessels branching at acute angles run into it at various parts, giving a salmon colour in the more acute stages. Some of these vessels remain after the affection has subsided, and form a valuable sign of its previous existence. Ciliary congestion accompanies this disease, and may be very intense. In most cases patients thus affected are the subjects of hereditary syphilis.

Dotted Opacity of the cornea (**Keratitis Punctata**) is found in many cases of serous iritis, serous cyclitis, and sympathetic ophthalmitis. The lower part of the cornea becomes hazy, and the seat of small dotted opacities, generally of greyish colour, but they may be white or pigmented. They are often arranged in the form of a triangle with the apex upwards. They are formed by the deposition of lymph on the posterior surface of the cornea. The anterior chamber is often increased in depth.

Onyx and Hypopyon sometimes accompany ulcers of the cornea and iritis. Onyx denotes a deposit of pus in the substance of the cornea. Hypopyon is an accumulation of pus in the anterior chamber. The pus lies at the

lowest part of the chamber, and its upper limit is horizontal and straight, or nearly so.

Ulceration of the Cornea is recognised by the loss of substance which it occasions. With this are generally associated more or less ciliary congestion, loss of transparency of the cornea, due to infiltration with inflammatory products, pain, blepharospasm, and contraction of the pupil.

The chief points to be noticed are the position, size, shape, number, amount and colour of the surrounding infiltration, the amount of ciliary congestion, and the presence or absence of vessels running to the ulcer. A large proportion of corneal ulcers is of merely local origin, and occurs as the result of slight injury, with or without subsequent infection. They often indicate a condition of impaired general health. The following are the principal ulcers:—

The **Simple Ulcer**, occurring as a small shallow depression with greyish floor and but little infiltration of the edges. The **Crescentic or Ring Ulcer**, which appears in the situation of or on the arcus senilis, and may entirely surround the cornea. The **Suppurating Ulcer**, a deep ulcer, situated near the centre of the cornea, with a yellow purulent deposit on its floor, and often accompanied by purulent infiltration of the cornea and hypopyon. The **Serpiginous Ulcer** also occurs near the centre of the cornea. The floor of this ulcer is greyish, and deeper towards the advancing edge, which is curved, raised, and of a yellowish-white colour. It is often complicated by hypopyon. The **Dendriform Ulcer**, which appears as a fine groove, throwing off lateral branches and having hardly any infiltration.

One very definite form of ulcer is due to **exposure of the cornea** to drying, dust and other irritants, owing to insufficient closure of the eyelids. It occurs as a dry-looking, grey depression on the lower part of the cornea, where it is not covered by the upper lid, and consequently has the shape of a crescent or of the segment of a circle. The chief diseases in which it occurs are:—Paralysis of the facial nerve, extreme cases of exophthalmic goitre, and in the last stages of exhausting diseases, such as infantile diarrhœa, cholera, malignant disease and meningitis. In meningitis, ulceration may perhaps be of neuropathic origin.

Neuropathic Ulceration of the cornea is characterised by rapid and general infiltration, giving it the appearance of moist wash-leather, the accompanying ciliary injection and other signs of inflammation being only slightly marked. It may be observed in paralysis of the ophthalmic division of the fifth nerve: in herpes zoster affecting the region supplied by this division, especially if the nasal branch is implicated, or even in the absence of any skin affection.

Phlyctenular Ophthalmia occurs in persons of the tubercular diathesis. In this affection small, greyish or yellowish elevations, accompanied by local congestion, appear at or near the margin of the cornea. These burst, leaving small round ulcers with yellow, infiltrated bases and edges, which show a tendency to advance towards the centre of the cornea, their track being marked by a leash of vessels from the conjunctiva. They often make their first appearance after an attack of measles or whooping-cough.

Ulcers are also left as a result of **herpes** of the cornea. This may occur in the course of herpes febrilis or of other diseases accompanied by herpes, such as pneumonia, typhoid fever and intermittent fever. Clear elevated vesicles appear, without vascularisation of the cornea. These burst, leaving a shallow irregular abrasion with a festooned edge. The corneal ulceration

of **small-pox** perhaps deserves separate mention. It appears about the tenth to the fourteenth day of the disease, after the secondary fever. It results from the spreading of an ulcer from a conjunctival pustule. Small-pox pustules do not present themselves on the cornea itself.

Affections of the Iris.—In examining the iris the chief points to be noticed are its colour and the size, shape and mobility of the pupil as compared with that of the opposite side. The depth of the anterior chamber and the ocular tension are also of importance in this connection.

This subject will be dealt with under the following heads :—

Iritis.
The results of Iritis.
Deformities of the Iris.
New growths of the Iris are so rare that they need not be described in this book.

Iritis.—There are two chief varieties of iritis, the plastic and the serous. In both the colour of the iris is altered, its lustre diminished, and its pattern obscured, so that it looks muddy. A blue eye becomes greenish and a brown one rusty. The pupil is more or less contracted, and its movements are sluggish. Ciliary injection (see p. 434) and a certain amount of conjunctival congestion, with blepharospasm and lacrimation, are generally present.

Plastic Iritis is characterised by the formation of lymph on the surface or in the substance of the iris and in the pupil. This gives rise to adhesions between the iris and the anterior capsule of the lens (posterior synechiæ). The presence of these is detected by the irregularity of pupil which they cause. This is rendered more obvious by dilating the pupil either by shading the eye or the use of atropine. The lymph may form yellowish or reddish nodules on the surface of the iris, especially in the later stages of secondary syphilis.

In **Serous Iritis** the exudation is chiefly fluid, slightly turbid from the presence of small particles of lymph. The accumulation of the fluid causes deepening of the anterior chamber and increase of ocular tension. Lymph is deposited as fine yellowish or greyish dots on a triangular area at the lower part of the cornea, keratitis punctata (see p. 435). Contraction of the pupil and ciliary congestion are only slightly marked.

Iritis may be of purely local origin, but the commonest cause of this affection is syphilis in its secondary stage. Many cases of chronic iritis are due to the rheumatic and some to the gouty diathesis. Iritis also occurs in small-pox (in the desquamative stage), typhoid fever, pneumonia, diabetes and septicæmia.

Results of Iritis.—As a result of inflammation the iris may be left of a lighter colour than natural, or a patch of dark pigment may make its appearance, or portions of the iris may be left pale, thinned and atrophic. The presence of synechiæ, or of the spots of lymph and pigment which they leave on the lens-capsule, is proof of a past iritis. Synechiæ cause the pupil to be of irregular shape. It may become oval or notched, or have a crenated outline. If the whole of the posterior surface of the iris is adherent the condition is called *total posterior synechia*. If it is adherent throughout the circumference of the pupil only, circular synechia results and causes *exclusion of the*

pupil. Sometimes the pupillary area is covered with lymph which becomes organised; the pupil is then said to be *occluded.* The pupil, if *excluded* or *occluded*, may be round, but cannot be dilated by means of midriatics. Under these circumstances aqueous humour accumulates behind the iris, bulging it forward and making it funnel-shaped, at the same time making the anterior chamber shallow. Secondary glaucoma may result.

Deformities of the Iris and Pupil.—The iris and pupil may be of abnormal shape from other causes than those mentioned in the preceding paragraph. As an effect of injury the iris may be separated from its attachment to the ciliary body for part of its circumference (irido-dialysis), or a portion of it may be folded back on itself (retroflexion of the iris). Occasionally the pupil is eccentric in position congenitally. Coloboma is a deficiency of the iris, generally at its lower and inner part, due to imperfect closure of the

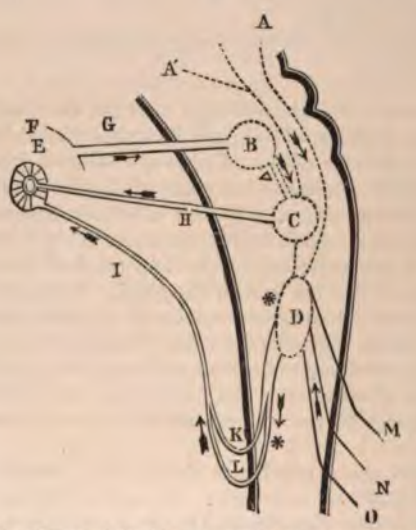


FIG. 238.—Scheme of the Nerves of the Iris. A, psychical impression; B, centrum optici; C, oculomotor centre; D, dilator centre (spinal); E, iris; G, optic nerve; H, oculomotor (sphincter) roots; I, sympathetic (dilator); K, L, anterior roots; M, N, O, posterior roots; Δ, seat of lesion causing reflex pupillary immobility; *, probable seat of lesion causing myosis. (*After Erb.*)

choroidal fissure in the fœtus. Fine threads attached to the anterior surface of the iris sometimes run across the pupil. They are remains of the fœtal pupillary membrane.

Disorders of Muscular Action.

(1.) **Paralysis of the Internal Muscles.**—The internal muscles of the eye are the ciliary muscle, the sphincter of the iris, and the dilator of the iris.

(*a.*) **Cycloplegia.**—Paralysis of the ciliary muscle, loss of accommodation. In this condition distant vision is good, but near vision is defective. Bilateral cycloplegia is one of the earliest and most common

symptoms of diphtheritic paralysis; it occurs also in locomotor ataxia and in cases of poisoning by belladonna. It may be due to disease of the anterior parts of the nuclei of the third nerves, or of the nerves themselves.

(*b.*) **Iridoplegia,** or paralysis of the iris. There are three varieties of paralysis.

i. *Loss of the reflex to light*, called reflex iridoplegia. In examining this reflex each eye must be tested separately, the other eye being covered. The patient should look at a distant object, while a bright light is brought suddenly in front of the eye.

ii. *Loss of the skin reflex.* Dilatation of the pupil may be produced

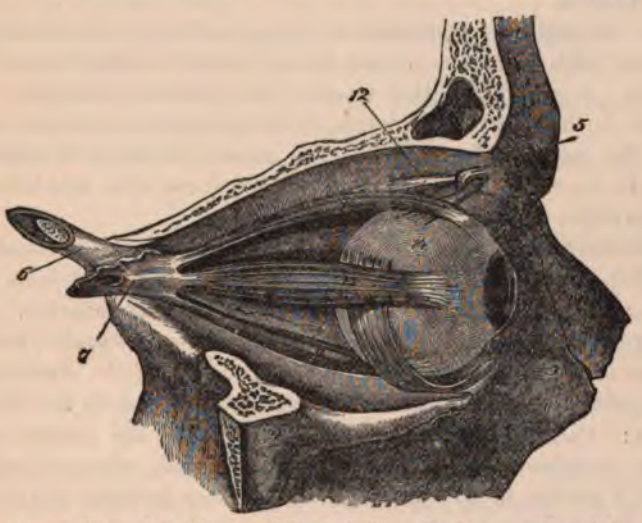


FIG. 239.—Lateral View of the Muscles of the Eyeball. 5, Trochlea or pulley of the superior oblique muscle, 12; 6, optic nerve; 8, superior; 9, inferior; and 12, external rectus; 13, inferior oblique.

in most healthy persons by stimulation of the cervical sympathetic by pinching or faradising the skin of the neck. This reflex is lost in some cases of damage to the cervical spinal cord or the cervical sympathetic. The skin reflex and the light reflex are often lost together, as in tabes, and in general paralysis of the insane. The light reflex is lost when there is atrophy of the optic nerves, or complete paralysis of the third nerves. It is maintained in blindness of central origin and in unilateral optic atrophy.

iii. *Accommodation iridoplegia.* The pupil does not contract when the patient looks from a distant to a near object. The condition is usually associated with cycloplegia, as in diphtheritic paralysis, but may exist alone. Loss of the reflex to light, but contraction of the

pupil in accommodation (the Argyll Robertson pupil), is a common symptom of locomotor ataxia.

iv. *Paralysis of the dilator.* This is generally one-sided, and is caused by pressure or disease of the cervical sympathetic (see p. 41). The affected pupil is smaller than the other, and does not dilate when shaded. The condition is associated with retraction of the eyeball, and slight narrowing of the palpebral fissure, but the movements of the upper eyelid remain normal.

(2.) **Paralysis of the External Muscles of the Eyeball.**—Paralysis of any of these muscles is mainly recognised by the following symptoms:—Defect of ocular movement, strabismus and diplopia or double vision.

The **limitation of movement** is brought out when the patient tries to follow with his eyes an object moved in various directions in front of him. It constitutes what is called the "primary deviation;" it is always in the direction of action of the paralysed muscle, and varies with the amount of paralysis. The term "secondary deviation" is given to the excessive movement of the sound eye when this is covered, and an object is fixed by the affected eye.

Strabismus, or the want of correspondence between the visual axes, is called convergent when the prolonged axes of the eyeballs would cross, divergent when the axes diverge from one another. Thus paralysis of one external rectus produces a convergent squint, paralysis of one internal rectus a divergent squint. Paralytic strabismus is distinguished from strabismus due to muscular spasm by the following points:—Paralytic squint is only present when the position of the object necessitates action of the affected muscle, whereas spasmodic squint is present in all positions, and secondary deviation of the sound eye does not occur.

Diplopia, common in paralytic squint, is usually absent in the spasmodic variety. Of the two images of an object looked at by a patient suffering from diplopia, the true one is seen by the sound eye, and is sharper in outline and more distinct than the false image, which is seen by the affected eye. Diplopia is said to be *simple* or *homonymous* when the false image is displaced towards the side of the paralysed eye; *crossed* when it is displaced towards the side of the non-paralysed eye. The former occurs with convergent, the latter with divergent strabismus.

In testing the eyes for diplopia, it is best to place a coloured glass before one of them while the patient looks at a candle-flame held in different parts of the field of vision.

The following is a summary of the chief symptoms which would result from *paralysis of each muscle of the left eye*:—

Sixth Nerve.—External Rectus.—Defect of movement outwards with convergent strabismus and diplopia on looking to the left. The images are vertical and parallel, the false image being to the left of the true one, and not tilted, unless the eyes look at an object above or below the horizontal level. The head is turned towards the left side.

Third Nerve.—Internal Rectus.—Defect of inward movement, divergent strabismus and crossed diplopia on looking to the right; the images are side by side, the false image being displaced in a horizontal line to the patient's right.

Superior Rectus.—Defect of movement upwards, and on trying to look upwards the left eye is rotated upwards and to the left by the action of the inferior oblique. The diplopia is crossed; the false image is higher than the true one, and is tilted towards the right.

Inferior Rectus.—Downward movement of the eyeball defective. On looking downwards, there is crossed diplopia, the false image being lower than the true one, and tilted to the patient's left.

Inferior Oblique.—On looking up, the left eye shows defective move-

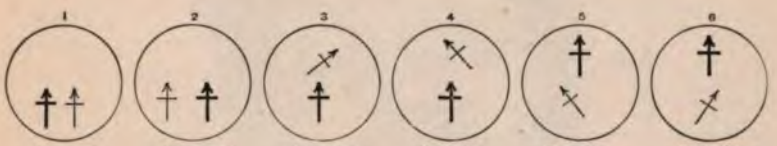


FIG. 240.—The Black Cross represents the *True Image*, the Thin Cross the *False Image*. The left eye is affected in all cases. (*After Bristowe.*)

ment, and its pupil is directed towards the right. The diplopia is simple; the false image is higher than the true one, and is tilted towards the left. The head is inclined backwards, and the chin turned a little towards the right side.

Fourth Nerve.—Superior Oblique.—On looking down, the left eye shows defective movement, and its pupil is directed towards the right. The diplopia is simple; the false image is below the true one, and is tilted towards the right.

Isolated paralysis of the external rectus is not uncommon, that of the superior oblique is occasionally met with, but the other muscles which are all supplied by the third nerve are usually paralysed together. In complete paralysis of the third nerve there is loss of the inward, upward, and, to some extent, of the downward movement of the eyeball; there is external squint, and the eye can only be moved outwards by the external rectus, and downwards and outwards by the superior oblique. There is also *ptosis*, or drooping of the upper lid from paralysis of the levator palpebræ; the pupil is moderately dilated, and

does not contract to light, and the power of accommodation is lost in consequence of paralysis of the ciliary muscle.

Paralysis of the third, fourth, or sixth nerve suggests disease at the base of the brain, in the sphenoidal fissure, or in the orbit. Paralysis of one third nerve may also be due to a lesion of the crus, and then there is hemiplegia on the opposite side of the body. Paralysis of the sixth in association with paralysis of the fifth or seventh nerve indicates disease of the pons. Paralysis of both third nerves suggests a tumour in the interpeduncular space, but this is a very rare condition.

Sometimes the movements of the eye rather than its individual

FIG. 241.—Total Ophthalmoplegia; Double Ptosis; Partial Paralysis of Face and Tongue; Optic Discs normal, and Fields of Vision unimpaired, so far as could be ascertained.

muscles are paralysed. Thus there may be loss of the upward movement of the eyes, in association with drooping of the upper lids (see Fig. 18, page 55); loss of convergence, either by itself or in combination with loss of accommodation; paralysis of lateral movement causing what is called conjugate deviation of the eyes.

These conditions are due to lesions of the nerve nuclei, or of higher centres in the cerebrum.

Conjugate Deviation of the Eyes with Turning of the Head may be due to paralysis or spasm. When due to paralysis, the head and eyes turn towards the side of the lesion if this is situated in a cerebral

hemisphere, but away from it when the lesion involves one side of the pons. The reverse is the case when conjugate deviation is produced by spasm in consequence of an irritative lesion in the pons or cerebrum.

Total Ophthalmoplegia means paralysis of all the ocular muscles; when this is complete the eyes are motionless, and, as partial ptosis is also frequently present, the patient has a peculiar sleepy expression (see Fig. 241).

External ophthalmoplegia means paralysis of the external muscles, *internal ophthalmoplegia* paralysis of the pupil and ciliary muscle. Each form is usually due to syphilis, and may occur alone or in association with hemiplegia, or with symptoms of locomotor ataxia. As a rule the lesion consists of a chronic degeneration of the oculo-motor nuclei. It is of great interest to observe the weakness of the orbicularis palpebrarum which frequently accompanies ophthalmoplegia, the association being analogous to that between paralysis of the lower facial muscles and the tongue in cases of bulbar paralysis.

Nystagmus.—This term is applied to involuntary rhythmical movements of the eyes, which are generally bilateral and symmetrical. The movements may be constant during waking hours, or may occur only when the eyes are moved in a particular direction. In the latter case weakness of certain muscles is indicated; for example, if horizontal twitching movements of the left eye are observed when a patient tries to look as far as possible to the left, it is probable that there is partial paralysis of the left external rectus.

Nystagmus occurs in many diseases of the nervous system, and is common in disseminated sclerosis, in Friedreich's disease, and in cases of tumours of the cerebellum.

It is frequently found in association with local affections of the eye —opacities of the cornea, diseases of the retina or choroid, &c.—which cause serious defect of sight. It is common in albinism. Sometimes it develops in adult life, apart from other evidence of disease, as in coal-miners.

Amblyopia and Functional Disorders of Sight.—The defects of vision to be considered here are—

Amblyopia and amaurosis.
Night-blindness.
Day-blindness.
Hemianopsia.
Coloured vision.
Micropsia.
Diplopia, not due to muscular derangement.
Colour-blindness.

The clinical examination of these affections involves the testing of

the acuteness of vision, the extent of the visual field, and the colour-sense.

Acuteness of vision is estimated by the patient's ability to recognise certain standard letters at a given distance. (For details, refer to text-books on diseases of the eye.) If vision is very defective, it is sufficient to note whether the outspread fingers can be counted at a short distance.

The extent of the visual field can be measured with sufficient accuracy for most clinical purposes, as follows:—Stand about two feet in front of the patient, tell him to cover one eye, and to look

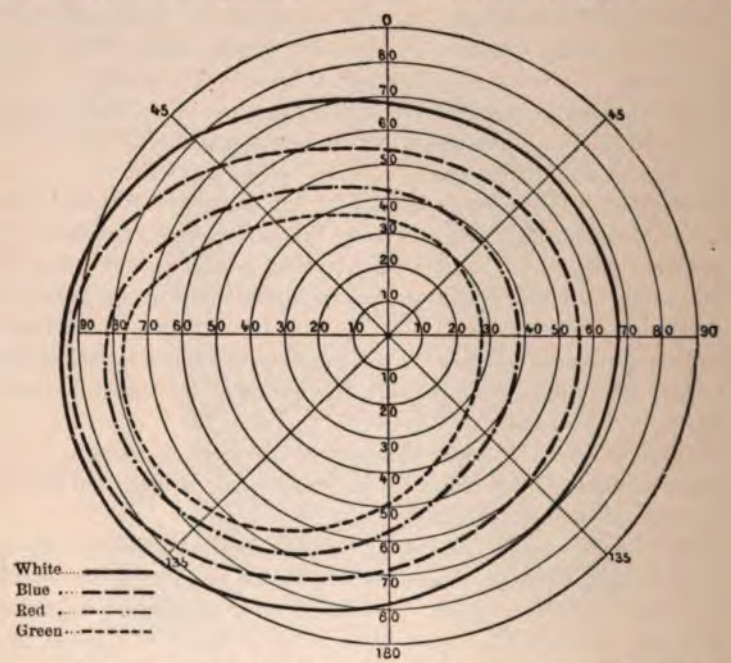


FIG. 242.—Field of Vision of Left Eye, showing Boundary Lines for Different Colours. (*Meyer.*)

steadily with the other at your nose. Then hold up a finger in a plane with your face and at some distance from it. Gradually bring this finger nearer to your nose and note the distance at which he begins to see it. Do this from various sides, and from above and below. Contraction of the field from one or from all sides may thus be detected; as also may central scotomata for red and green, if small pieces of paper of these colours be used in place of the finger-tip.

In order to obtain a correct map of the field of vision it is necessary to use the "Perimeter," an instrument in which an arm shaped like a quadrant of a circle, and graduated in degrees, moves round a central

pivot on which the patient's eye is fixed. Then a white or coloured object is moved along the arm, which is placed at various angles, and the points at which the object ceases to be seen mark the limits of the field. The results are recorded on prepared charts. In a normal eye the field for the object is larger than that for colour, while the fields for colour diminish in the following order:—White, blue, yellow, red, green, violet (see Fig. 242). As a rule, it is sufficient to test the visual field for red and green.

Colour perception is best tested by getting the patient to match a skein of wool of a certain colour with all the skeins of a similar colour which are present in a collection of skeins of every colour and shade. It is also useful to ask the patient to identify and name certain colours, but it must be remembered that a patient may not know the names of certain colours, or, while unable to perceive them, he may give the correct names.

Amblyopia means diminished acuity of vision, accompanied by no abnormal ophthalmoscopic appearances, or only by slight signs of optic neuritis or atrophy. When the dulness of sight amounts to actual blindness it is called amaurosis. It may be due to disease or functional disturbance of the retina, optic nerve, optic tract, or visual centre. It may be symmetrical or asymmetrical.

(1.) *Amblyopia from suppression of image* ("congenital amblyopia") is the defect of vision often present in the squinting eye of children, with convergent strabismus associated with error of refraction (hypermetropia). The defect is chiefly noticeable in that part of the field of vision which is common to both eyes.

(2.) *Amblyopia from defective retinal images* ("amblyopia ex anopsia"). In cases of very high hypermetropia, or astigmatism, where clear images have never been formed on the retina, full optical correction of the error of refraction may fail to raise the vision to the normal standard.

(3.) *A rarer form of asymmetrical amblyopia*, coming on somewhat rapidly, and involving chiefly the central region, and perhaps associated with slight haziness and congestion of the optic disc, may be due to exposure to cold, or may be of reflex origin from irritation of the fifth nerve, as by carious teeth.

(4.) *Toxic amblyopia* is generally caused by the abuse of tobacco. It is progressive and symmetrical. The discs are slightly congested and hazy in the early stages, pale and somewhat atrophied in the later. The defect is most marked in the central part of the field. Very often there is a central scotoma for red and green.

In diabetes mellitus a central or peripheral defect of vision may be present, and sometimes optic atrophy is found. Uræmic amblyopia, or, more commonly, amaurosis, occurs suddenly in some cases of puerperal eclampsia and scarlatinal nephritis.

(5.) *Severe hæmorrhage*, as from the stomach, bowels, or uterus, may cause blindness either immediately, from defective blood-supply to the retina and visual centre, or later possibly from optic neuritis.

(6.) *Concentric restriction of the field* occurs in atrophy of the optic nerves; it is also a frequent symptom in cases of hysteria. In hysterical amblyopia the contraction of the field is most extreme on the anæsthetic side of the body, and affects colour vision; the other eye may also have its field reduced in size to a variable extent, but colour vision is unaffected. Associated with the amblyopia there is

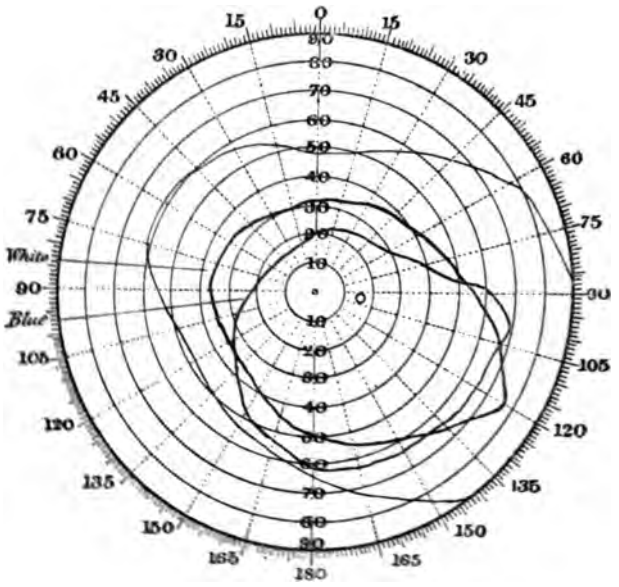


FIG. 243. Perimetric Tracing of the Field of Vision for Colours in the Right Eye, from a Case of Paralysis (multiple neuritis) due to poisoning by Carbon Bisulphide. The fields of vision were much contracted in each eye for white and blue, while those for red and green were absent. The optic discs were paler than natural. Almost complete recovery a month later. (*Ross and Bury*, p. 122.)

often some degree of *asthenopia*, in which there is weakness of the ciliary muscle and of the internal recti, together with photophobia, and it will sometimes be found that owing to speedy weariness of the eye the first perimetric chart shows a larger field than a second one taken immediately afterwards.

A similar "crossed amblyopia" is occasionally met with as a symptom of cortical disease, probably from damage to the supramarginal and angular convolutions: the dimness of sight affects the eye on the side opposite to the cerebral lesion.

Night-blindness (nyctalopia) signifies much diminished acuity of vision in dim light. It is a symptom of retinitis pigmentosa, and is frequently prominent in cases of syphilitic retinitis. It may also be congenital, and

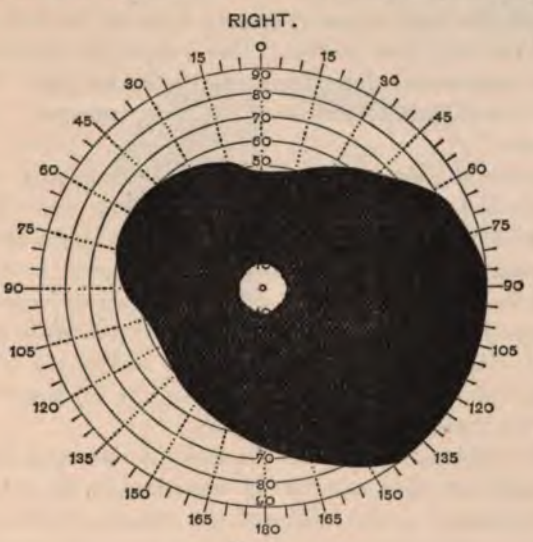


FIG. 244.

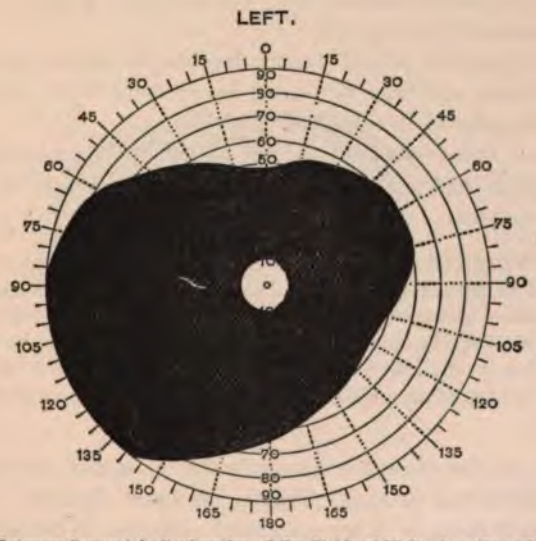


FIG. 245.—Extreme Concentric Contraction of the Fields of Vision in a Case of "hysterical amblyopia," and right hemianæsthesia.

quite unaccompanied by ophthalmoscopic changes. Acute nyctalopia occurs after exposure to very bright lights, and is often associated with scurvy.

Day-blindness occurs in some cases of congenital amblyopia.

Hemianopsia means loss of one-half of the visual field, not due to intra-ocular disease. The line separating the lost from the retained half-field is generally vertical, and deviates in the middle, so as not to interfere with the fixation point: it may, however, be oblique, or only a sector of the field may be lost. Hemianopsia is absolute when all three visual sensations (colour, form, and light) are lost. If only one (colour) or two (colour and form) are lost, it is "relative." Most cases are absolute.

Homonymous or lateral hemianopsia is the commonest variety, the right or left half of the fields of both eyes being lost. Loss of the right half-fields implies loss of function of the left halves of the retinæ, and *vice versâ*. Homonymous hemianopsia may occur as a functional disturbance in migraine, but as a rule it depends on organic disease, and the lesion implicates either (1) some part of the visual path behind the chiasma, viz., the optic tract, the hinder end of the internal capsule, or the white fibres of the occipital lobe; or (2) the visual centre itself, viz., the cortex of the occipital lobe.

Temporal hemianopsia, due to blindness of the nasal half of each retina, is a rare affection, and may be unnoticed by the patient. It is produced by damage to the middle of the chiasma, which involves the crossed fibres, as from the pressure of a tumour or a distended third ventricle, or a localised meningitis. The symptom is met with in some cases of acromegaly (see p. 33).

Nasal hemianopsia, a still rarer affection, is due to injury of the uncrossed fibres, and may be caused by pressure on or inflammation involving the sides of the chiasma.

Altitudinal hemianopsia means loss of the upper or lower halves of the visual fields, and would indicate a lesion of the upper or the lower part of the chiasma.

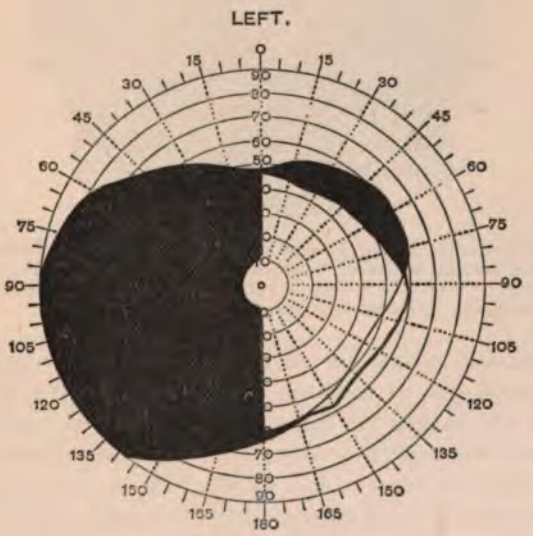
Coloured Vision (chromatopsia) is not uncommon after extraction of senile cataract, and in exhausted states of the system. It is also an effect of the administration of santonin. Red vision is most commonly complained of.

Micropsia.—Objects appear smaller than they really are in cases where accommodation is only effected by excessive effort. This disorder of vision is sometimes the aura of an epileptic attack. It is a common symptom in syphilitic retinitis.

Diplopia (double vision) is generally due to squint, and has already been considered (see p. 440). This is binocular diplopia. Uniocular (or monocular) diplopia is an occasional symptom in hysteria, dislocation of the lens, irido-dialysis, commencing cataract and astigmatism, and may occur as a result of the instillation of eserine or atropine.

Colour-blindness (achromatopsia) may be observed in tobacco-amblyopia, in optic atrophy after neuritis, and in optic atrophy associated

FIG. 246.



F.G. 247.

Fields of Vision in a case of left-sided Hemianopsia. The black areas represent the blind parts. The patient also suffers from slight left hemiplegia and partial left hemianæsthesia and the lesion—probably softening from syphilitic thrombosis of the lenticulo-optic artery—most likely involves the hinder end of the right internal capsule. Note (1) that the line of separation between the blind and seeing halves deviates at the fixation point, leaving central vision intact; and (2) that there is a slight restriction of the right halves of the fields of vision.

with diseases of the spinal cord, especially locomotor ataxia. It occurs also in hysterical amblyopia, and in some diseases of the retina.

Changes detected by means of the Ophthalmoscope.—A knowledge of the use of the ophthalmoscope should be acquired at an early period of the student's career, so that he may soon become familiar with the leading aspects of the fundus in health and disease; for a detailed account of these we must refer him to special works, it is only possible in the present manual to give a brief summary of some of the chief morbid changes.

Disease of the Choroid.—The common changes met with are of an atrophic character, that is, pale or white patches with rings or spots of brown or black pigment in the neighbourhood.

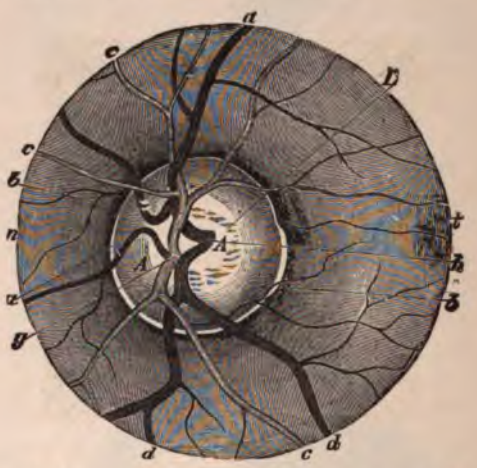


FIG. 248.—The Entrance of the Optic Nerve, with the adjacent parts of the Fundus of the Normal Eye. *a*, ring of connective tissue; *b*, choroidal ring; *c*, arteries; *d*, veins; *g*, division of the central artery; *h*, division of the central vein; L, lamina cribrosa; *t*, temporal (outer) side; *n*, nasal (inner) side. (*Landois and Stirling.*)

In *Choroiditis disseminata* discrete or confluent atrophic areas are found scattered over the fundus. The condition occurs most frequently in syphilis, and in both the hereditary and acquired forms at a period varying from six months to three years from the commencement. It may be limited to one eye, but usually both eyes are affected. The change is commonly most marked at the periphery. White patches in the central region also occur in myopia, and in elderly persons, often as a result of hæmorrhages. Large atrophic patches are sometimes seen in hydrocephalus, and may follow the absorption of tubercular masses in the choroid.

Tubercles in the Choroid must be carefully distinguished from the white spots of atrophy; they are more yellowish in tint, have a rounded

form, and pigmentary disturbance is usually absent. They are often present in cases of tubercular meningitis, but may exist in acute miliary tuberculosis, apart from meningitis.

Diseases of the Retina.—When inflamed (retinitis) the retina loses its transparency and appears hazy or smoky; the opacity is either diffuse or it occurs in spots and patches. *Pigmentary deposits* in the retina are arranged in a lace-like or reticulated form, or in sharply defined lines, and not in blotches and rings, as in the choroid. They are superficial to the vessels.

Syphilitic Retinitis, in which there are diffuse hazy opacities, a blurred disc, and tortuous vessels, occurs as a secondary symptom in both congenital and acquired syphilis. Its onset is commonly rapid, its course

FIG. 249.—Atrophy after Syphilitic Choroiditis, showing various degrees of wasting. *a*, atrophy of pigment epithelium; *b*, atrophy of epithelium and chorio-capillaris, the large vessels exposed; *c*, spots of complete atrophy, many with pigment accumulation. (*After Hutchinson and Nettleship.*)

chronic, and failure of sight is a conspicuous feature. As a rule it is secondary to choroiditis.

Albuminuric Retinitis is characterised by the presence of (1) a greyish haze, (2) sharply defined opaque white dots or patches, (3) hæmorrhages, and (4) inflammation of the disc (papillitis). One or other of these changes predominates, and they are variously combined in different cases. The condition is nearly always significant of a chronic nephritis, and occurs most commonly in granular kidney; it is often a marked feature in the albuminuria of pregnancy.

Hæmorrhages in the retina, of various shapes and sizes, are frequently met with in leucocythæmia and pernicious anæmia; they are also sometimes present in cases of scurvy, purpura, ague, septicæmia, and ulcerative endocarditis; and they usually form a part of every severe retinitis

or papillitis. Retinitis hæmorrhagica, a condition in which numerous small linear or flame-shaped hæmorrhages are scattered over the fundus, is occasionally seen in gouty persons, and in the subjects of arterial degeneration.

Retinitis pigmentosa is a chronic symmetrical disease which tends to atrophy of the retina, with much pigmentary deposit and secondary atrophy of the disc. The disease is hereditary, and begins in early life; contraction of the visual fields soon occurs, and blindness ultimately results.

Embolism of the Central Artery of the retina produces instantaneous blindness of the affected eye. The central region looks misty, and the opacity is greatest around the yellow spot; the vessels, especially the arteries, are diminished in size; white atrophy of the disc supervenes,

FIG. 250.—Recent severe Retinitis in Renal Disease. (*After Gowers and Nettleship.*)

and sight becomes impaired or is completely lost. Embolism of this artery may take place at the same time as embolism of a cerebral artery.

Optic Neuritis or Papillitis consists in swelling and increased vascularity of the optic papilla or disc. The edge of the disc becomes blurred, and the retinal veins enlarged; the swelling of the disc renders it unduly prominent, and the vessels bend down abruptly at its edge; the opacity extends, and appears to enlarge the disc; the veins become broader and the arteries narrower, and blood patches may be visible on the surface or margins of the swollen area. Such changes may disappear almost entirely, but commonly the disc passes into a state of "consecutive" atrophy, presenting a staring white colour and sharply cut edges. Care must be taken not to mistake haziness of the disc, often seen in cases of hypermetropia, for true neuritis.

Double Papillitis, not always equally marked in the two eyes, is

strongly suggestive of intracranial disease, and especially of tumours of the brain. It is also frequently met with in tubercular basal meningitis, but only very rarely in cases of cerebral hæmorrhage or of thrombotic softening. It has sometimes been observed in cases of embolic softening, and occasionally in diffuse cerebritis; also in multiple sclerosis, and rarely in chronic myelitis. Papillitis with or without retinitis also occurs in albuminuria, lead poisoning, simple anæmia, and after the various exanthemata, especially scarlet fever and typhoid.

Unilateral Papillitis is usually due to disease at the back of the orbit or near the optic foramen, as from the local pressure of a tumour.

Disturbance of sight may be absent even when there is considerable swelling of the discs, and when present is often more marked during

FIG. 251.—Severe recent Papillitis; several elongated patches of blood near the border of the disc. (*After Hughlings Jackson and Nettleship.*)

the subsidence than during the active period of the inflammation. Failure of vision, occurring early and soon becoming extreme, points to local pressure on the chiasma, as from a distended third ventricle in hydrocephalus, a new growth, or a local meningitis. In such cases the pupils are dilated and immovable, whereas in neuritis from a central lesion they usually preserve their reaction to light.

Atrophy of the Optic Nerve is either secondary, following a severe papillitis or embolism of the central artery of the retina; or primary, when it results either from pressure on some part of the nerve or chiasma, or in consequence of a chronic sclerosis of the nerve fibres. The most frequent cause of such sclerosis is locomotor ataxia, and after this multiple sclerosis. In locomotor ataxia, optic atrophy may precede all other symptoms by several years.

DISORDERS OF HEARING.

Tests.—1. "Air conduction" is conveniently tested by means of a watch held at varying distances from one ear, while the other is closed by the finger. The distance at which the ticking just ceases to be audible is measured and compared with that of the opposite ear, or, if this is diseased, with an ear of average acuteness.

2. "Bone conduction." Normally a watch held close to but not touching the closed ear is nearly or quite inaudible, while if it be applied to the skull near the ear, the ticking is loudly heard. Disease

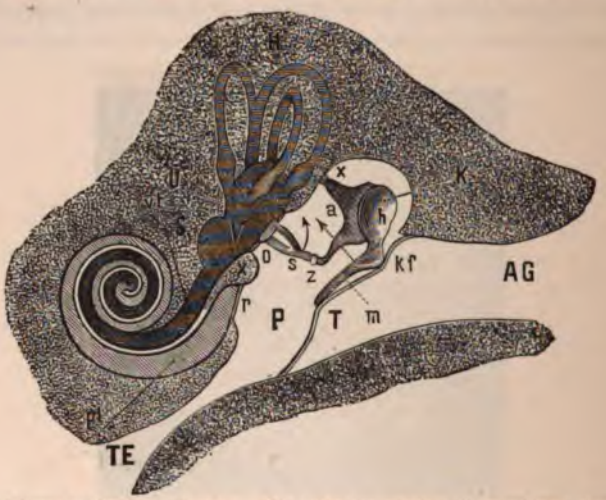


FIG. 252.—Scheme of Organ of Hearing. *AG*, outer ear meatus; *T*, membrana tympani; *K*, malleus, with its head (*h*), short process (*kf*), and handle (*m*); *a*, incus; *x*, its short leg; *s*, stapes; *z*, Sylvian ossicle; *P*, middle ear; *o*, fenestra ovalis; *r*, fenestra rotunda; *x*, beginning of cochlea; *pt*, scala tympani; *vt*, scala vestibuli; *V*, vestibule; *S*, saccule; *U*, utricle; *H*, semicircular canals; *TE*, Eustachian tube. The long arrow shows the line of traction of the tensor tympani; the short curved arrow that of the stapedius. (*Macalister.*)

of the external or middle ear produces the same result as closure of the passage with the finger; but in disease of the auditory nerve or of the labyrinth, both air and bone conduction are impaired.

Deafness.—(1.) Apart from local changes in the external auditory meatus, in the Eustachian tube, or in the tympanum, the most frequent cause of deafness is disease of the labyrinth, or of the nerve endings themselves. These parts are affected in old age, in some of the acute specific fevers, especially mumps, and in congenital syphilis. In Menière's disease, deafness is associated with vertigo and vomiting.

(2.) Deafness from intracranial disease is commonly caused by a lesion of the auditory nerve at the base of the brain, as from syphilitic

meningitis. It may also result from damage to the auditory nuclei within the pons, or from a lesion above the nuclei, affecting the tegmentum, or the internal capsule, or the upper part of the temporo-sphenoidal lobe.

(3.) Loss of hearing may occur in hysteria or in anæmia. Sometimes it follows the administration of quinine or the salicylates.

Subjective Noises, Tinnitus Aurium, may be produced by obstructions or disease in any part of the ear, and rarely by lesions affecting the nerve, its nucleus, or the auditory path in some part of its central course.

"Buzzing" or "roaring" sounds are common in anæmia. Sounds like bells or music are generally of central origin, and occasionally constitute an epileptic aura. A pulsating sound may be due to intracranial aneurysm, and in rare cases a murmur may be heard on applying the stethoscope to the posterior part of the skull.

DISORDERS OF TASTE.

Tests.—The front of the tongue, the back of the tongue, and the palate should be separately tested with sugar, quinine, salt and dilute acetic acid; the two first-named substances are best appreciated at the back, the two latter at the tip and edges of the tongue.

Ageusis,—loss or diminution of the sense of taste,—affects one half of the tongue in cases of hemianæsthesia, whether of organic or of functional origin. Unilateral ageusis of the anterior portion of the tongue may occur from damage to the chorda tympani, to the facial nerve between the chorda tympani and the geniculate ganglion, to the second branch of the fifth, to its roots, or to the Gasserian ganglion. The path of taste from the palate and back of the tongue, according to Gowers, is along the glossopharyngeal nerve, tympanic nerve, and small petrosal to the otic ganglion, and thence along the third division of the fifth nerve to its root. Lesions of the root of the fifth nerve have abolished taste on the corresponding side of the tongue and palate.

Perversion of the sense of taste—**parageusia**, or increased sensation of taste—**hypergeusia** or subjective taste sensations, may each occur in hysteria and in insanity, may result from ear disease, or constitute the aura of an epileptic attack.

DISORDERS OF SMELL.

Tests.—1. Such substances as camphor, musk, oil of cloves, valerian, assafœtida, which affect the olfactory nerves alone, should be applied first to one nostril, then to the other; but bodies like ammonia or acetic

acid, which irritate the branches of the fifth nerve, should not be employed. 2. Flavour may be tested by holding in the mouth cheese or wine.

Anosmia,—loss or diminution of the sense of smell,—is most commonly due to local disease of the nose, as from polypi. It may also occur:—As a congenital affection; in old age; in paralysis of the fifth nerve, and sometimes in paralysis of the seventh nerve; from injury or disease of the olfactory nerves, bulbs, or tracts; in thrombosis or embolism of the anterior cerebral artery; sometimes in locomotor ataxia; also in association with, and on the same side as hemi-anæsthesia, whether of functional or organic origin.

Hyperosmia, or increased sensitiveness of the olfactory nerves, is occasionally present in cases of hysteria and insanity. More frequently in hysteria, certain odours produce great disgust, headache, or fainting.

Hallucinations and **illusions of smell**, such as subjective odours of sulphur, or of putrid bodies, are met with in the insane: they may occur as auræ of epileptic fits; and are occasional symptoms of an intracranial tumour.

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